ASSESSMENT OF COPD

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OBJECTIVES

The general objective of this chapter is to assist physicians and other allied health care professionals from the hospital and community settings identify and assess the patient's chronic obstructive pulmonary disease (COPD) and subsequent severity stratification by implementation of a thoughtful approach to spirometry. This approach will be based on an assessment of the magnitude of underdiagnosis, the potential effectiveness of intervention, the predictive value of spirometry, and the clinical profile of patients who may have COPD. After reading this chapter, the physician and the allied health care professional will be able to

- improve the early detection of COPD diagnosis based on specific clinical indicators;
- understand the limits of history, physical examination, and radiology in the diagnosis of COPD;
- consider adopting spirometry as the gold standard for diagnosis of airway obstruction;
- differentiate between spirometry and other pulmonary function tests;
- consider the merits of adopting spirometry screening of "at-risk" patients in primary care;
- refer to specific professionals when needed.

Recently published consensus guidelines for the management of asthma and COPD outline markedly different therapeutic approaches to these overtly similar obstructive airway diseases. Chronic obstructive pulmonary disease may be considered as the "forgotten illness" since so much public awareness and educational initiatives have been instead focused around asthma management.

Chronic obstructive pulmonary disease, which represents a mixture of pathologic processes, should not be defined based on symptoms because it is nonspecific, nor should it be based on a pathologic definition because this is impractical in clinical practice. The diagnosis is usually made relatively late in the natural history; it is frequently made after the age of 40 years, when symptoms have been in place for a long time, when the patient is restricted from performing certain activities, or, even worse, when this impairment or disability limits fulfilment of a normal role at work and/or at home.

Confirming the diagnosis requires spirometry. Although spirometry is the "gold standard" for diagnosis and disease severity stratification, this tool still remains underused outside specialist practice; consequently, a significant number of individuals remain unidentified. The debate on spirometry as a screening tool used to assist in diagnosis has been raised and dropped on various occasions over the last 30 years.^{1,2}

This chapter will explore failings in history recordings, physical examination, and radiologic assessment of patients with suspected COPD; furthermore, it will extol the value of office spirometry over the inappropriate reliance on radiology and will highlight spirometry as an essential screening tool rather than a laborious ritual.

DEFINITION OF COPD

Chronic obstructive pulmonary disease is synonymous with chronic airflow limitation, chronic obstructive lung disease, and chronic airflow obstruction. Chronic obstructive pulmonary disease is characterized by a single physiologic feature, which is the limitation of expiratory airflow owing to obstruction of the airway. This airflow limitation is generally progressive; it may be accompanied by airway hyperreactivity and may also show partial reversibility in response to pharmacologic agents.

Two major pathologic subtypes, chronic bronchitis and emphysema, are embraced under the rubric "COPD" and may be present independently or together, depending on the case. Although not always possible, it is clinically worthwhile to separate asthma from COPD. However, we have to bear in mind that obstructions in many patients with COPD can include a significant and reversible component; moreover, some patients with asthma may go on to develop irreversible airflow obstruction that is indistinguishable from COPD. Other conditions that may lead to the limitation of expiratory airflow, such as upper airway obstruction, bronchiectasis, sarcoidosis, cystic fibrosis, or bronchiolitis obliterans, are usually excluded from the definition.

Chronic bronchitis is defined as a clinical disorder characterized by excessive mucus secretion. It is manifested by a chronic or recurrent productive cough on most days, for a minimum of 3 months in a year and for more than 2 successive years. It is important to remember that not all patients with chronic bronchitis will have airflow obstruction since chronic bronchitis is defined by history, not by spirometry.

In contrast to the clinical description of chronic bronchitis, emphysema has long been defined in anatomic and pathologic terms alone. If a clinical definition of emphysema did exist, it would include the presence of effort dyspnea, which is a nonspecific symptom.

PATHOPHYSIOLOGIC CHANGES

Pathologic changes in the lung lead to corresponding physiologic changes. Mucus hypersecretion in the airways leads to chronic cough and sputum production, which is characteristic of chronic bronchitis. Although these symptoms can be present for years before other symptoms or physiologic abnormalities of airway obstruction develop, they do not necessarily mean that the patient has an airway obstruction.

In COPD, a variety of pathophysiologic mechanisms can contribute to varying degrees of airflow obstruction. Although contraction of airway smooth muscle is generally regarded as an important mechanism underlying airflow obstruction in asthma, it can also play a role in patients with COPD. This overlap between asthma and COPD has led to some confusion in the clinical classification of patients. Part of the confusion is attributable to the underuse of confirmatory spirometry in primary care practice or the limited access to hospital spirometry for primary care physicians. There are other more important mechanisms independent of smooth muscle contraction that are responsible for airflow obstruction in COPD. Fixed airway obstruction is believed to be primarily owing to fibrosis and alteration in small airway structures. Emphysema, which results in the destruction of alveolar attachments and inhibits the ability of the small airways to maintain patency, plays a smaller role. In addition, patients with COPD can have altered production and clearance of secretions and varying degrees of airway inflammation. All of these contribute to reduced airway caliber and airflow obstruction, especially during exacerbation.³

DIAGNOSTIC STRATEGIES

It is important to make a timely diagnosis of COPD to ensure proper triage and appropriate treatment. The most pressing requirement is to be aware of COPD. A firm diagnosis can be made only by an objective assessment of airway obstruction through the use of spirometry. A diagnosis of COPD should be considered in patients who have (1) a history of progressive and persistent dyspnea on exercise, with or without chronic and productive sputum, and (2) previous or current exposure to noxious particles or gases such as tobacco smoke or occupational dust and chemicals.

Patient Medical History

A focused patient history is always a good beginning. Table 2–1 presents some "tips and tricks" to direct a history taking in patients with suspected COPD. Because of the large reserves in lung function and the slowly progressive nature of COPD, patient symptoms, and thus a clinical diagnosis, are often delayed until extensive damage has occurred. Dyspnea is the main symptom that brings the patient to medical attention. It progressively results in decreased exercise tolerance and thus reduces activities of daily living. Increased or chronic sputum production is often present, although some patients may complain only of dyspnea. Other symptoms, such as wheezing and chest congestion or tightness, might be present but are relatively nonspecific. It is important to realize that because many patients have been living with their symptoms for such a long time, they have accordingly learned to adjust to their limitations and may minimize their complaints of dyspnea, cough, and even disability. These symptoms may remain undetected for some time unless the physician proceeds systematically with a careful medical history. In addition to the medical history being specific to the respiratory condition, the physician should also assess the following:

1. Past medical history, childhood illnesses, occupational exposure, etc

- Presence of comorbid conditions (heart diseases, respiratory diseases such as asthma at a young age, gastroesophageal reflux, chronic sinusitis, obstructive sleep apnea)
- 3. Impact of disease on work and activities of daily living
- 4. Family and social support

Although certain elements in the patient's history might seem directly diagnostic of asthma, this is not always the case. These misleading elements can include intermittent symptoms with what appear to be a normal respiratory condition between attacks, asthma at a young age, allergic rhinitis, and/or atopy. Chronic obstructive pulmonary dissease can be associated with all of these symptoms and may coexist with asthma.

Physical Examination

Although a physical examination is an essential part of patient assessment, it is a crude and insensitive means of detecting airflow limitation. Regrettably, undergraduate medical education and postgraduate train-

To Be Assessed	Specific Areas to Be Addressed		
Smoking habits	Record smoking of cigarette, cigar, or pipe Age patient started to smoke with number of cigarettes smoked per day and estimation of total "pack-years"* of smoking Current smoking status; if patient is an ex-smoker, include date patient stopped smoking; if not, was there any attempt to discontinue smoking in the past		
Environment with specific attention to occupational exposure	Ensure a chronologic review Specific environmental and work exposure		
Cough	Frequency and duration: intermittent, every day (seldom, only nocturnal) Nature of cough: productive or nonproductive (especially on awakening)		
Dyspnea	Progressive and present every day Presence of labored breathing: exercise related, resulting in progressive activity limitation Precipitants other than exercise, especially respiratory infection Perceived severity of dyspnea (ie, activity level/limitation)		
Wheezing	Frequency and duration, diurnal pattern Factors precipitating		
Acute respiratory infections	Frequency and timing Presence of cough, dyspnea, sputum and sputum purulence, wheezing, and fever Requiring treatment such as antibiotics or systemic corticosteroids; requiring physician visits, emergency department, and hospitalization		

TABLE 2-1 Assessment of Patient Respiratory History

*Number of pack-years = number of packs of cigarettes/day multiplied by number of years of smoking (eg, one pack of 20 cigarettes smoked per day for 1 year = one pack-year).

ing programs continue to emphasize the bedside approach to early detection of disease. This misplaced trust in the physical examination is most evident in recent articles addressing the clinical utility of physical examination. Holleman and Simel reviewed the clinical examination and its ability to predict airflow limitation, examining some 44 articles in the field.⁴ Most physical findings considered to represent airflow limitation or hyperinflation showed low levels of agreement among observers. Physicians seldom agree on the absence of the apical pulse, whether a patient has a subxiphoid apical impulse, and, finally, the presence of hyperresonance and the degree of diaphragmatic excursion. Agreement is only slightly increased when clinicians attempt to determine the presence or absence of wheezing or the intensity of breath sounds. Given that these are best-case scenarios, with selected physicians performing under ideal test conditions, it is questionable whether under realistic clinical conditions, the usual physical examination can offer any useful information. Holleman and Simel reported that surrogate measures of airflow by physical examination means are more likely to be agreed on by physicians.⁴ Specifically, they recommend the "match test" or the measurement of forced expiratory time. The match test requires patients to extinguish a lighted match held 10 cm from the open mouth. Failure to do so is associated with a "higher likelihood of airflow limitation" being present, but the measurement of forced expiratory time has not been standardized. Apparently, the measurement is most precise and useful when multiple expiratory efforts are made and when a stopwatch is used by the physician.

Holleman and Simel offered the inevitable conclusion that "no single item or combination of items from the clinical examination rules out airflow limitation." However, they failed to move to the next inescapable conclusion: objective measurements of airflow are necessary. Instead, they offered a complex scheme of risk factors and physical findings to estimate the risk that airflow limitation is present. Presumably, a moderate or high likelihood of the presence of airflow limitation would lead to the use of spirometry to quantify the severity of the defect. If the diagnosis is suspected from symptoms and/ or high-risk predisposing factors, spirometry should be ordered regardless of the physical findings. The purpose of prolonged and imprecise physical examination, requiring multiple expiratory maneuvers, stopwatches, and lighted candles, must then be questioned. Can such an approach be cost effective?

It appears, however, that the physical examination remains important to assess alternative diagnoses or associated comorbid conditions that are common in patients with COPD. In acute exacerbation of COPD, there are also important changes on physical examination. One example that should bring the patient to immediate medical attention is the presence of any change in the patient's alertness.

Radiologic Studies

Although it is known to be an insensitive means in detecting airflow obstruction and hyperinflation, the standard posteroanterior and lateral chest radiographs are often performed during the work-up of a patient with suspected COPD. Although the chest radiograph of the patient with mild COPD is likely to be normal, that of a patient with advanced disease may feature a flattened diaphragm, increased retrosternal air space, and apparent hyperlucency, usually considered characteristic of emphysema. Similarly, thickened bronchial walls may be seen in chronic bronchitis. The presence of bullae is strongly suggestive of emphysema. Chest radiographs are primarily useful in identifying or ruling out alternative diagnoses or associated comorbid conditions such as lung cancer. This is especially true in patients with symptoms of acute exacerbation, which could be mimicked by other acute diseases such as pneumonia, pneumothorax, pulmonary edema, or pulmonary emboli.

Computed tomography (CT) of the chest, particularly when using high-resolution techniques, is more useful to make a diagnosis of emphysema but should be reserved for the work-up of patients with only particular interventions in mind. More specifically, the CT scan can be used to detect and quantify the severity of emphysema in patients with decreased carbon monoxide diffusion, increased lung volumes, and/ or impaired gas exchange. Thurlbeck and Muller reviewed the pathologic and CT scan findings of emphysema and noted that significant statistical correlations have been found between the two.5 In moderate to severe emphysema, the severity may be underestimated by CT scan findings. Nonetheless, the CT scan remains the best way to recognize emphysema in living patients. It is not indicated for routine clinical use but is helpful in the investigation of patients with emphysema if bullectomy or lung volume reduction surgery is being considered. The

CT scan can detect nonhomogeneity in the degree of emphysema and can give some indication of the probable success of resectional surgery, especially when correlated with ventilation/perfusion lung scans. Furthermore, it may be useful in the assessment of patients who have dyspnea and reduced diffusion capacity without evidence of airflow obstruction. There are reports of such patients who have CT scan findings of emphysema. Stern and Frank reported that a CT scan might be as "sensitive as pulmonary functions tests at detecting emphysema, and is more sensitive than the standard chest radiography (96% vs. 68%)."⁶ The CT scan can detect regional emphysema, whereas spirometry proves a global indication of lung function.

MEASUREMENT OF AIRFLOW OBSTRUCTION

Peak Expiratory Flow and Spirometry Measurements

Peak Expiratory Flow

The use of peak expiratory flow (PEF) rate measurements has been advocated in the management of asthma but has no utility in managing COPD. Advocates of the physical examination have recently adopted PEF measurements in their quest for indirect measurements that might obviate the need for spirometry or at least help select only high-risk individuals for whom spirometry would be requested.

In COPD, there is a poor relationship between PEF and forced expiratory volume in 1 second (FEV₁), and it is impossible to predict FEV₁ from the PEF or vice versa.⁷ Peak expiratory flow may underestimate the degree of airway obstruction in COPD⁷ and is relatively insensitive to obstruction of the small airways (mild or early obstruction). Moreover, PEF is very dependent on patient effort and has about twice the inter- and intrasubject variability with mechanical PEF meters, which are much less accurate than spirometers.⁷

Spirometry

Spirometry is neither invasive nor hazardous and should not be expensive. This simple measurement procedure should be readily available to assess possible airflow limitation in regular smokers and in those with chronic or recurrent respiratory symp-

toms, occupational exposure, or family history. The family physician is in an ideal position to identify the patients at risk because of the long-term relationship and knowledge of the patient's lifestyle and medical history. As an example, consider the patient with comorbid ischemic heart disease who presents to his/her family doctor with increasing dyspnea. That person may have quit smoking years ago before his/her coronary history and worsening of dyspnea was thought to be cardiac in origin, but no one has thought to perform spirometry. That can be very helpful to diagnose airway obstruction instead of focusing on ischemic heart disease as the primary cause of the dyspnea. All too often, patients with chronic dyspnea and comorbid diseases have never had spirometry performed as outpatients or even perioperatively.

Why do primary care physicians underuse spirometry? There are many potential reasons to consider:

- Lack of understanding/knowledge because of the lack of education in medical schools. Many family physicians have graduated without developing a working knowledge of spirometry.
- Many offices do not have the equipment, time, or staff requirements to adequately perform or interpret spirometry. This requires an effort to develop easy accessibility to spirometry testing and incorporate it into office protocols.
- 3. Some family physicians have a negative attitude toward COPD as they are used to diagnosing it only in the late/advanced stages, when therapeutic modalities are of less benefit. Earlier diagnosis and intervention in milder disease must be encouraged.
- 4. Family physicians are not using spirometry enough for monitoring ongoing treatments and disease follow-up.

Spirometry requires no more effort on the part of the patient than the forced expiratory time measurement recommended by Holleman and Simel.⁴ Moreover, reliable volume time and flow volume spirometers have become available at an ever-decreasing cost in the past several years. Given the prevalence of both asthma and COPD, it would seem reasonable for any primary care clinician to have easy access to a spirometer, as they do for an electrocardiogram. Spirometry testing requires some skill and quality control (which is easily learned) to ensure accurate and reproducible testing. Simple guidelines have been set out for performing spirometry by the American Thoracic Society (ATS) Snowbird workshop.⁸

Further Lung Function Assessment

Chronic obstructive pulmonary disease and asthma may be characterized by reduced FEV₁, a reduced ratio of FEV1 to forced vital capacity (FVC) (FEV₁/FVC \leq 70%), and gas trapping. Spirometry measurement cannot reliably distinguish between broad categories of obstructive airway disease. Measurements of bronchodilator response provide some useful distinguishing information. In the most straightforward example, a return to normal lung function following the inhalation of a β_2 agonist is most compatible with asthma and would rule out COPD. When the postbronchodilator FEV_1 remains subnormal, both asthma and COPD are diagnostic possibilities. Various schemes have been used to qualify the bronchodilator response in an effort to offer diagnostically useful information. The most common rule of thumb is that a response of 15% and 180 cc above baseline indicates significant bronchodilator reversibility and a diagnosis of asthma. Yet, such an approach tends to overestimate the responsiveness of subjects with low baseline FEV₁, so that subjects with severe emphysema will paradoxically appear to have the greatest bronchodilator reversibility. A more reasonable approach would be to express bronchodilator responses as a percentage of the predicted normal value. However, it still seems uncertain as to what degree of reversibility will effectively make one patient asthmatic and another a victim to COPD.

A reduction in diffusion capacity is characteristic of emphysema and may help distinguish it from asthma or chronic bronchitis. Given that COPD is often an admixture of chronic bronchitis and emphysema, any reduction in diffusion capacity is more suggestive of COPD than asthma. This laboratory-based study might be a useful baseline measurement for primary care physicians attempting to make initial diagnostic distinctions. However, it is important to remember that patients with COPD might not have a reduction in diffusion capacity.

OFFICE SPIROMETRY VERSUS LABORATORY PULMONARY FUNCTION

Spirometry should follow the patient history and the physical examination. Other tests of airway function may be performed and should be requested as needed by the physician. It is important, however, to differentiate the need for office spirometry from the more labor-intensive full pulmonary function testing. This testing includes spirometry, assessment of lung volumes and diffusion capacity, and gas exchange (ie, oxygen saturation and sometimes arterial blood gases), all of which are usually performed in the pulmonary function laboratory at a local hospital. Postbronchodilator FEV₁ is the best single measurement to follow a patient's therapy for outpatient/office follow-up assessment and adjustment of medications and treatment (Table 2–2).

STAGING COPD SPIROMETRY AND CLINICAL PRESENTATION

The approach to COPD would be greatly facilitated by the implementation of a staging system that would allow standardization of patients with COPD. Currently, there is no staging system for disease severity that would combine symptoms, impairment in airflow, or functional impairment and health status. Unfortunately, there are also no data defining their interrelationship in a qualitative manner. A decreased FEV1 correlates best with increased mortality and morbidity. Consequently, COPD severity is still staged on the degree of the airflow obstruction. According to the criteria of the ATS statement on the interpretation of lung function, stage I is $FEV_1 \ge 50\%$ of predicted, stage II is FEV_1 35 to 49% of predicted, and stage III is FEV_1 < 35% of predicted.³ However, as shown in Table 2-3, there is no universal agreement on these FEV₁ cutoff levels between different organizations, that is, the European Respiratory Society (ERS),⁹ the British Thoracic Society (BTS),7 and the new Global Initiative for Chronic Obstructive Lung Disease (GOLD) guidelines.¹⁰ Chronic obstructive pulmonary disease describes a spectrum of disease progressing from the earliest symptomless stages through to respiratory failure. The medical requirement of patients increases with increasing severity. Chronic obstructive pulmonary disease has been divided into three stages that approximate to the health care requirements of the patients at each stage. The precise boundaries chosen differ among the ATS,3 ERS,9 BTS,7 and the ever-evolving GOLD guidelines.¹⁰ It is important to remember that, at best, there is only a modest relationship between FEV1 and dyspnea, functional impairment,

Laboratory Test	Purpose of Test		
Spirometry (pre-/postbronchodilator)	Essential for confirming the presence and possible reversibility of airflow obstruction and quantifying airflow obstruction		
Pulmonary function test Lung volumes	Measurements other than forced vital capacity may be necessary in moderate to severe cases (eg, presence of giant bullae, mixed ventilatory limitation, etc)		
Carbon monoxide Diffusion capacity (DLLO)	In circumstances such as disproportional dyspnea in relation to seriousness of airflow obstruction or in emphysema		
Arterial blood gases	Necessary in advanced COPD (FEV ₁ < 35% of predicted value or with clinical signs suggestive of respiratory failure or right-sided heart failure) In cases of serious airflow obstruction or during acute deterioration, follow-up measurements are important		

TABLE 2-2 Spirometry and Laboratory Function Testing

COPD = chronic obstructive pulmonary disease; FEV1 = forced expiratory volume in 1 second.

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	FEV_1 Predicted of Normal Value (%)			
Classification of disease severity	ATS ³	BTS ⁷	ERS ⁹	GOLD ¹⁰
Stage I (mild)	≥ 50	60–79	≥70	≥ 80
Stage II (moderate)	35–49	40-59	50–69	30-80
Stage III (severe)	< 35	< 40	< 50	< 30

TABLE 2-3 Staging Chronic Obstructive Pulmonary Disease for Disease Severity*

*In all patients with a reduced FEV1/FVC ratio, usually less than 70%, which is the mark of obstructive ventilatory impairment.

 FEV_1 = forced expiratory volume in 1 second; FVC = forced vital capacity; ATS = American Thoracic Society; BTS = British Thoracic Society; ERS = European Respiratory Society; GOLD = Global Initiative for Chronic Obstructive Lung Disease.

and health-related quality of life. Disease severity staging based on spirometry value should be regarded only as a general indication of the approach to management. Some patients with stage III lung function (FEV₁ < 35% of predicted) may be relatively asymptomatic with a sedentary lifestyle, whereas other patients will be very symptomatic, with lesser degrees of airway obstruction because of their more active lifestyle. Family physicians tend to adjust therapy according to the portrayed symptoms. It is also well known that all-cause mortality, including coronary heart disease and stroke, increases with decreasing FEV₁.¹¹

Mild COPD

In patients with mild COPD, there are often no symptoms and no abnormal physical signs. Morning cough and sputum or breathlessness on vigorous exertion should alert the health professional to the possibility of COPD. Clinical examination and investigation other than pulmonary function tests are likely to be normal in these patients. Often, it is just the history of smoking or other potential lung injury (ie, occupational) that leads to the early diagnosis of COPD. A firm diagnosis will be made only by an objective assessment of airway obstruction through the use of spirometry.

Moderate COPD

In patients with moderate COPD, a wide range of symptoms can be present, although there are usually only a few clinical signs. The patients may have cough and sputum production and sometimes wheezing or breathlessness on moderate exertion such as physical work or climbing hills. Acute worsening of symptoms may be associated with an acute exacerbation. Clinical examination may appear normal, but there may be wheezes, rhonchi, or reduced breath sounds. Investigations such as chest radiography are useful to rule out other diseases such as pneumonia or cancer. Once again, a firm diagnosis will be made only by an objective assessment of airway obstruction through the use of spirometry.

Severe COPD

In patients with severe COPD, a progressive disabling breathlessness on minimal exertion or at rest is present. However, the individual patient's perception of breathlessness can vary considerably for the same degree of airflow obstruction.¹²

Cough, sputum, and wheezing can also be present. Complications may develop, such as cor pulmonale (right-sided heart failure) with cyanosis and peripheral edema. Clinical examination is not always diagnostic, but often there are signs of chronic hyperinflation (loss of cardiac dullness, hyperresonance to percussion, increase of the anteroposterior diameter of the chest). Pursed-lip breathing, use of accessory respiratory muscles, and retraction of the lower intercostal muscles are present in severe COPD. When it is very severe, there may be weight loss, central cyanosis, signs of hypercapnia (flapping tremor, drowsiness), raised jugular venous pressure, and peripheral edema. These signs may not be present in stable severe COPD and may occur only during acute exacerbation. Investigations with chest radiography will show increased bronchial markings and/or hyperinflation. Hypoxemia and hypercapnia may also be seen, but as mentioned previously, it is possible to make a firm diagnosis only through the use of spirometry.

SPIROMETRY SCREENING

Some experts recommend spirometry screening to prevent underdiagnosis and maximize the opportunity to intervene, as is done with hypertension.^{1,2} However, mass screening remains controversial because it may result in overdiagnosis and overuse of health resources. Although spirometry results have been shown to correlate with the development of COPD in men who smoke, only a similar correlation has been variably demonstrated in women.¹³

"Normal" readings in smokers may cause undue complacency, whereas "abnormal" spirometry readings, based on predicted ideal values, may cause undue alarm. Finally, even aggressive intervention is unlikely to produce a high smoking cessation rate, and the correlation between screening and smoking cessation remains uncertain.¹³ Spirometry is well established as a necessary diagnostic tool.^{3,7,14} It also demonstrates and reinforces the doctor's diagnosis and treatment regimen. A thoughtful approach to spirometry screening should include assessment of the magnitude of underdiagnosis, the potential effectiveness of intervention, the predictive accuracy of spirometry, and, finally, an overview of the clinical profile of COPD.

Evidence of Underdiagnosis

It has long been believed that COPD is underdiagnosed and that there appear to be several contributing factors. The first factor is based on the similarity of symptoms between COPD and chronic asthma, which makes them clinically indistinguishable from each other. Many asthmatics smoke or have smoked but are not necessarily smoke susceptible; many patients with COPD have hyperreactive airways but are not necessarily asthmatic. Several studies have also demonstrated that asthma, like COPD, is associated with an accelerated decline in lung function, which is irrespective of smoking status.

The most likely cause of underdiagnosis, however, is that disabling COPD symptoms do not appear until the disease is well advanced and pulmonary function is significantly impaired. Wolkove and coworkers have shown that statistically significant changes in FEV₁ do not necessarily represent important differences in symptoms for patients.¹² These authors also demonstrated that the correlation between acute changes in spirometry values and dyspnea is, in fact, very weak.¹² Thus, COPD patients with an FEV₁ that is significantly lower than the predicted normal value may not perceive symptoms related to airway limitation. Conversely, most asthmatic patients will perceive their symptoms related to the degree of airway obstruction much sooner. It is quite usual for patients to come to the physician's attention in clinic only when their FEV_1 is lower than 50% of predicted normal value.

Effectiveness of Intervention

Smoking is by far the most significant risk factor for COPD, and its effects are potentiated by age. As described by Fletcher and colleagues,¹⁵ FEV₁ in susceptible smokers does not begin to diverge signifi-

cantly from the normal range of decline until about age 35, typically after 20 years or more of smoking, but then it falls precipitously compared with normal values (the "horse-racing effect"). Fletcher and Peto have developed this horse-racing hypothesis, suggesting that those with the worst function (FEV₁) at a single observation have deteriorated most rapidly prior to measurement and will continue to do so.^{16,17}

The Lung Health Study was a triumph in its demonstration of the benefits of smoking cessation in patients with COPD.^{13,18,19} However, it did not show that spirometry results themselves are a significant motivating factor in sustained smoking cessation, nor did it conversely evaluate whether the finding of normal spirometric values might encourage smokers in their habit. Moreover, the Lung Health Study's limited objective of screening smokers over the age of 35 years did not afford an opportunity for assessing the social usefulness of mass spirometry screening in the general population at risk. Many studies have shown that the single most effective intervention to help smoking cessation is the influence of the family physician; although unproven in practice, the identification of abnormal spirometry and realization of the consequence should be a powerful motivation for both physician and patient toward smoking cessation and lifestyle modification.

Introducing spirometry into a primary care setting can lead to profound changes in both diagnosis and treatment. One study in primary care found that using spirometry to screen at-risk patients led to a new diagnosis of COPD in 17% of patients, whereas 18% of patients with a previous diagnosis of COPD had normal spirometry and were misdiagnosed. Information from spirometry led to a change in therapeutic regimens in 37% of patients.²⁰

Matrix for Spirometry Screening

The Lung Health Study demonstrated that spirometry screening in selected populations is an effective way to identify individuals at risk for COPD and to initiate appropriate interventions. Approximately 15 to 20% of smokers have a rapid decline in FEV₁ and need to be identified for COPD. This is a very large detection rate for a screening test in primary care compared with many screening procedures. Effective interventions, such as smoking cessation, can lead to major improvements in morbidity and mortality.

In 1983, the ATS issued a position statement that discouraged population screening for COPD.²¹

It was felt that a positive "one-to-one" correlation between decreased lung function in smokers and future development of COPD had not been adequately demonstrated. A perfect correlation is rare in preventive medicine, but it can be argued that the association between decreased lung function and smoking established in the Lung Health Study is as sound as that between coronary artery disease and hypertension. The second objection claimed that the screening test must be able to detect disease at a point at which effective intervention can affect the outcome. Again, the resounding success of smoking intervention in mild COPD in the Lung Health Study should adequately address any such concerns.

The topic of spirometry screening for COPD was revised in a consensus statement from the National Lung Health Education Program 2000, which recommends widespread use of office spirometry by primary care providers for smoking patients over 45 years old. Spirometry was also recommend for patients with respiratory symptoms such as chronic cough, episodic wheezing, and exertional dyspnea to detect airway obstruction owing to asthma or COPD. Women represent a potential "tidal wave" of new cases of COPD.²²

Should routine screening for COPD be considered in primary care with spirometry? Based on the literature, a reasonable approach for surveillance spirometry of the general population at risk is to screen all smokers over the age of 35 or any patients with respiratory symptoms. If this model appears too one-dimensional, COPD screening can also be conceived of in the context of a matrix that shifts through a few straightforward risk factors in connection with the complex pathophysiology of the disease. Smoking is the major risk factor for COPD, but only a small percentage of smokers are at risk.^{21,23} Age is a factor, but significant airway limitation may also be present in younger, asymptomatic patients. Methacholine reactivity is a factor, but it is linked to smoking and airflow limitation and does not necessarily indicate an asthmatic constitution. Similarly, occupational and environmental irritants may be risk factors, particularly in individuals who smoke. Target populations for whom spirometry screening is recommended are listed in Table 2-4.1 In any situation, an underdiagnosis of COPD represents a missed opportunity to permanently intervene. If the results of recently published studies do not address the issue

TABLE 2–4 Target Populations for Spirometry Screening*

- All smokers 35 years of age and over
- Current or past smokers with a 20 pack-year history of smoking, whether or not the patient complains of
 respiratory symptoms
- · Patients with recurrent or chronic respiratory symptoms including cough and breathlessness on exertion
- · Patients who have significant occupational exposure to respiratory irritants
- · Patients with a family history of obstructive pulmonary disease
- Patients with a history of hyperresponsiveness to provocative agents
- Patients with childhood factors that may be associated with the development of chronic obstructive pulmonary disease: low birth weight, prematurity, bronchopulmonary dysplasia, frequent respiratory infections, exposure to environmental tobacco smoke

*If initial spirometry is normal, repeat spirometry in 2 to 3 years or earlier if patient presents with respiratory symptoms.

of mass screening, they at least provide a compelling rationale for screening that is targeted to populations at risk.

FOLLOW-UP ASSESSMENT

After initial assessment of their COPD, patients are followed up in no systematic manner. The frequency of visits and the need for follow-up investigations vary widely between physicians. It is important to note that the follow-up for monitoring postbronchodilated FEV₁ is the best objective measure of lung function in COPD.

More recently, management of many patients with COPD can be best performed by case management coordinators. These are usually nurses or other health professionals who have additional training and expertise in the management of patients with COPD. They function best as important facilitators and patient advocates, aiding the seamless flow of information to and from the patient, family physician, and specialist, and can become important points of contact for exacerbations rather than the emergency department.

Pulmonary rehabilitation programs with exercise training are becoming more numerous and available to patients with COPD. Widespread availability of these programs in community hospitals, not only at academic centers, is to be encouraged.

Patients' self-directed management plans ("action plans"), which are widely adopted in the asthma population and clinics, are now being adapted for the patient with COPD.

WHEN TO REFER TO A RESPIROLOGIST

No evidence-based guidance can be provided as to which patients in the community should be referred for specific assessment and follow-up by a respirologist apart from unusual features being present. But a generalized description could include the following factors: age under 40 years, smoking history of less than 10 pack-years, when the diagnosis is in doubt, or for disabled patients. Patients with frequent attendance at the emergency department or who have been hospitalized with a COPD exacerbation are also worthy of a respirologist's assessment. Some areas require that patients needing home oxygen therapy be assessed by a respirologist.

SUMMARY

Many patients are troubled by dyspnea, cough, and/or sputum production. Both patients and physicians seem complacent with these symptoms. Although patients may consult physicians with acute episodes of cough and sputum, physicians can be lulled into a sense of security by the absence of textbook physical signs of advanced COPD. Ordering chest radiographs should be superseded by a thorough history and spirometry.

Spirometry is a simple to use, cost-effective instrument that primary care physicians should consider for their practice. Spirometry itself takes little time to perform and provides, as illustrated in this chapter, an objective measurement crucial to the appropriate diagnosis and severity stratification of patients with obstructive lung disease. Physicians should be just as comfortable with performing and interpreting spirometry as with blood pressure and electrocardiograms. Objective tests such as spirometry are significantly underused; therefore, many individuals with COPD remain undiagnosed and untreated.

To become proficient and knowledgeable about spirometry, various workshops have been developed, including full-day Mainpro C workshops endorsed by the College of Family Physicians of Canada. They usually have a significant component of hands-on experience and emphasize quality control issues, along with indications for spirometry and interpretation. These courses are very popular and well attended. A guide to the interpretation of spirometry in primary care, dually endorsed by the College of Family Physicians of Canada and the Canadian Thoracic Society, is available.²⁴

Health care workers must emphasize public health policies around smoking cessation; they must consider implementation of screening programs directed toward individuals in high-risk groups with spirometry and provide care in keeping with national guidelines. Spirometry should be easily accessible to patients in the primary care office or in local pulmonary function laboratories. Although we strongly support the widespread use of spirometry, it is important for physicians to order this test and not the full pulmonary function tests for assessment and follow-up.

CASE STUDY

Ms. Cope, a 54-year-old woman, presents at her annual physical examination.

Medical History, Physical Examination, and Test Results

Medical History

- She denies any significant respiratory complaints but does state that during the last three winters, her smoker's cough produced sputum on most days.
- She complains of fatigue, occasional cough, and shortness of breath on exertion essentially when she is trying to keep up with her daughter walking in the mall.
- She is limited in her activities of daily living; however, she states that she is not taking part in any sports.
- She is a current smoker and has smoked approximately half a pack per day for 26 years (13 packyears).
- She has no asthma, allergies, or gastrointestinal or cardiac symptoms.
- She has a family history as follows: her mother died aged 70 with stroke and her father died aged 67 with lung cancer (both were heavy smokers); one sister aged 42 has breathing difficulties and is taking inhalers.

Physical Examination

- There is no respiratory distress.
- She has a respiratory rate of 18 breaths per minute, temperature 37.2°C, heart rate 70 beats per minute, and blood pressure 140/80 mm Hg.
- She has a clear chest, some wheeze on forced expiration, a normal cardiac examination, and no peripheral edema or cynanosis.

Test Results

- The chest radiograph is normal.
- A blood test for α_1 -antitrypsin deficiency is performed because of the family history of a sister in her forties with breathing difficulties: 1.55 g/L (reference range 1.24–1.92 g/L).
- Spirometry is compatible with mild obstruction without significant reversibility (Table 2–5).

Questions and Discussion

Ms Cope has a history compatible with chronic bronchitis and spirometry in keeping with a clinical diagnosis of COPD. There is no history in keeping with asthma. She describes no intermittent episodes of dyspnea, cough, wheeze, or chest tightness with exertion or exposure to allergens. She has no posterior nasal drip or heartburn. There is no family history of asthma or childhood symptoms. This woman's history provides a classic description for diagnosis of chronic bronchitis, that is, daily cough productive of sputum on most days for 3 months for 2 consecutive years.

	Prebrond	Prebronchodilator		Postbronchodilator	
	Measurement	% Predicted	Measurement	% Change	
FVC	4.65 L	109	4.85 L	4	
FEV ₁	2.60 L	79	2.80 L	8	
FEV ₁ /FVC ratio		56			

TABLE 2-5 Spirometry Pre- and Postbronchodilators

FVC = forced vital capacity; FEV₁ = forced expiratory volume in 1 second.

In this case, physical examination would not allow such a certain clinical diagnosis. History and spirometry are the key items to appropriate diagnosis. Office spirometry confirms that she has significant obstruction (FEV₁/FVC ratio \leq 70%) and mild obstructive lung disease (FEV₁ of 79%). The bronchodilator response is not significant, which is in keeping with a diagnosis of COPD.

A chest radiograph is not diagnostic of COPD; however, it does help exclude an alternate diagnosis such as pneumonia, pulmonary fibrosis, and congestive heart failure if suspected clinically. The chest radiograph in this case was normal.

Blood tests are not helpful in making the diagnosis; however, because of the family history and young age of her sibling with respiratory symptoms, a blood test for α_1 -antitrypsin deficiency is recommended.

As her spirometry showed mild disease, further detailed pulmonary function tests such as lung vol-

KEY POINTS

- Physicians should have a high index of suspicion for the diagnosis of COPD.
- Chronic obstructive pulmonary disease may present insidiously and may be easily confused with disease in other organ systems; patients complaining of chronic respiratory symptoms, especially breathlessness on exertion, should have a spirometry for the purpose of diagnosis.
- Smoking cessation efforts in primary care, along with institution of spirometry screening of individuals in at-risk groups, is paramount.
- Physicians unfamiliar with indications for spirometry or performance and interpretation of spirometry may consider an update as a learning need.
- Public awareness and education campaigns are critical in their direction toward COPD diagnosis and therapy.

umes and diffusion, along with oxygen saturation or arterial blood gas determination, are not required.

Follow-up

Office follow-up assessment should include a history, assessment of functional status, and postbronchodilator FEV_{1}

Inhaler technique should be checked at each visit. Education and information on exercise, diet, and follow-up are important. The patient has only ceased smoking for only 3 months, and it is crucial to emphasize how important it is that she does not restart and to offer assistance if needed. An annual influenza immunization and consideration of a once in a lifetime Pneumovax vaccination, along with institution of bronchodilator therapy for symptom relief on exertion, can be discussed and are reviewed in greater detail elsewhere in this textbook.

• Specialist referral for confirmation of diagnosis should be easily available to patients with advanced disease, severe symptoms, or presentation at a young age.

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