Diagnosis of Chronic Obstructive Pulmonary Disease

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Chronic obstructive pulmonary disease affects more than 26 million adults in the United States. Family physicians provide care for most of these patients. Cigarette smoking is the leading risk factor for chronic obstructive pulmonary disease, although other risk factors, including occupational and environmental exposures, account for up to one in six cases. Patients presenting with chronic cough, increased sputum production, or progressive dyspnea should be evaluated for the disease. Asthma is the disease most often confused with chronic obstructive pulmonary disease. The diagnosis of chronic obstructive pulmonary disease is based on clinical suspicion and spirometry confirmation. A forced expiratory volume in one second/forced vital capacity ratio that is less than 70 percent, and that is incompletely reversible with the administration of an inhaled bronchodilator, suggests chronic obstructive pulmonary disease. Disease severity is classified by symptomatology and spirometry. Joint guidelines from the American Thoracic Society and the European Respiratory Society recommend a single quantitative test for alpha$_1$-antitrypsin deficiency in patients diagnosed with chronic obstructive pulmonary disease who remain symptomatic despite bronchodilator therapy. Other advanced testing is usually not necessary. *(Am Fam Physician.* **2008;78(1):87-92. Copyright © 2008 American Academy of Family Physicians.)**

▶ **Patient information:** A handout on this topic is available at http://familydoctor.org/online/famdocen/home/articles/706.html.

Chronic obstructive pulmonary disease (COPD) is the fourth leading cause of chronic disease–related morbidity and mortality in the United States, accounting for more than 120,000 deaths annually.[1] Mortality from COPD has increased substantially over the past 20 years,[2] and it is estimated that it will become the third leading cause of chronic disease–related morbidity and mortality in the United States by 2020.[3] The cost of caring for patients with COPD in the United States exceeds $37 billion annually.[4] Family physicians play a central role in the timely diagnosis of COPD. *Table 1* includes Web sites for more information about the disease.

Definition

COPD is defined as an inflammatory respiratory disease, largely caused by exposure to tobacco smoke. The disease is characterized by a progressive and incompletely reversible airflow obstruction. The key elements of COPD are exposure, primarily to cigarette smoke; airway inflammation; and airflow obstruction that is not fully reversible.[5] The diagnosis of COPD is based on signs and symptoms and is confirmed by spirometry. However, symptoms are underrecognized by patients, and COPD is underdiagnosed by physicians. It is important to note that the terms chronic bronchitis and emphysema are no longer included in the formal definition of COPD, although they are still used clinically.[5] Emphysema is a pathologic term used to describe destruction of the alveolar-capillary membrane. Chronic bronchitis is a clinical term used to describe the presence of cough or sputum production for at least a three month duration during two consecutive years.

Risk Factors

Cigarette smoking is the primary risk factor for COPD. More than 80 percent of deaths from the disease are directly attributable to smoking, and persons who smoke are 12 to 13 times more likely to die from COPD than nonsmokers.[6] The absolute risk of COPD among active, continuous smokers is at least 25 percent.[7]

SORT: KEY RECOMMENDATIONS FOR PRACTICE

Clinical recommendation	*Evidence rating*	*References*
COPD should be suspected in persons presenting with cough, dyspnea, or increased sputum production, especially those with a history of smoking.	C	1, 15
Joint ATS/ERS guidelines recommend screening for alpha$_1$-antitrypsin deficiency in symptomatic adults who have persistent obstruction on pulmonary function testing and in asymptomatic adults with a persistent obstruction on pulmonary function testing who also smoke or have a significant history of occupational exposure.	C	22
Spirometry confirms a COPD diagnosis.	C	5, 12, 20
Spirometry should be performed in patients 45 years or older who smoke and have a persistent cough.	C	26

ATS = American Thoracic Society; COPD = chronic obstructive pulmonary disease; ERS = European Respiratory Society.

A = consistent, good-quality patient-oriented evidence; B = inconsistent or limited-quality patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, see http://www.aafp.org/afpsort.xml.

However, other risk factors are responsible for an increasing number of COPD cases.[8] These risk factors include advancing age, secondhand smoke exposure, chronic exposure to environmental or occupational pollutants *(Table 2[9])*, alpha$_1$-antitrypsin deficiency, a childhood history of recurrent respiratory infections, and a family history of COPD.

Pathophysiology

The pathophysiology of COPD is related to chronic airway irritation, mucus production, and pulmonary scarring. Irritation from environmental pollutants (most commonly, cigarette smoke) or a genetic predisposition leads to airway inflammation, which causes increased mucus production and decreased mucociliary function. This combination of increased mucus and decreased mucociliary clearance leads to the hallmark COPD symptoms of coughing and sputum production. "Smoker's cough," an early-morning paroxysm of hacking cough to expectorate respiratory secretions, is also characterized by coughing and sputum production. Smoker's cough is often clinically referred to as chronic bronchitis.

Continued airway irritation and inflammation cause scarring within the airways. This leads to progressive airway obstruction and dyspnea, which prompts most patients to seek medical attention. Irritation, inflammation, mucus production, and scarring also predispose patients to respiratory infections, which is another common reason for patients to seek medical attention. Barring symptoms, many patients do not seek medical care; thus, the pathogenesis of COPD can progress for years before diagnosis or treatment.

Table 1. Web Sites for More Information About Chronic Obstructive Pulmonary Disease

American Thoracic Society
Web site: http://www.thoracic.org/sections/copd

Canadian Thoracic Society
Web site: http://www.copdguidelines.ca/home-accueil_e.php

Global Initiative for Chronic Obstructive Lung Disease
Web site: http://www.goldcopd.com

Institute of Clinical Systems Improvement
Web site: http://www.icsi.org/chronic_obstructive_pulmonary_disease_2286.html

U.K. National Institute for Health and Clinical Excellence
Web site: http://www.nice.org.uk/guidance/CG12

U.S. Department of Veterans Affairs
Web site: http://www.oqp.med.va.gov/cpg/COPD/COPD_base.htm

Epidemiology

Population-based statistics suggest that more than 10 million adults in the United States have been diagnosed with COPD.[2] However, the National Health and Nutrition Examination Survey (NHANES) suggests that roughly 10 percent of the adult U.S. population has evidence of impaired lung function consistent with COPD,[10] placing the number of adults with the disease closer to 26 million. This disparity supports the widely held belief that COPD is underdiagnosed.[11] As a direct response to the underdiagnosis of COPD, the general medical services contract in the United Kingdom now requires physicians in that country to use a formal COPD disease registry plus regular clinical spirometry when diagnosing the disease.[12]

Table 2. Occupational Exposures Associated with COPD

Mineral dust	
Coal mining, hard rock mining, tunnel work, concrete manufacturing, silica exposure	
Organic dust	
Cotton, flax, hemp, grains	
Noxious gas	
Sulfur dioxide, isocyanates, cadmium and other heavy metals, welding fumes	

NOTE: *Occupational exposure to dust as a risk factor for COPD is independent of cigarette smoking. The effects of cigarette smoking and occupational exposures are additive in terms of increasing the risk of COPD.*

COPD = chronic obstructive pulmonary disease.

Information from reference 9.

COPD is more common and more often fatal in women than in men. There are several reasons for this discrepancy. Because of differences in lung size and mechanics, women's airways are more hyperresponsive to exogenous irritants than are men's airways.[13] Therefore, women have a larger degree of lung damage and functional respiratory impairment for a given amount of tobacco or irritant exposure than men.[13] Although the diagnosis of COPD is often overlooked in both populations, it is diagnosed even less in women than in men.[14]

Clinical History

SYMPTOMS

A COPD diagnosis is based on clinical suspicion in patients presenting with any of the hallmark symptoms (i.e., cough, increased sputum production, and dyspnea), especially in patients with a smoking history.[1] One study found that patients with newly diagnosed COPD presented for medical care because of a concerning cough (85 percent), exertional dyspnea (70 percent), or increased sputum production (45 percent).[15] Wheezing has also been reported as an initial presenting symptom in up to 40 percent of patients with COPD.[10] However, the relationship between the degree of airflow obstruction and patient perception of symptoms is highly variable; some patients with advanced airflow limitation may be relatively asymptomatic. The NHANES survey showed that only 60 percent of patients with moderately reduced forced expiratory volume in one second (FEV_1; 50 to 85 percent of predicted) complained of symptoms.[10] Less commonly reported symptoms associated with COPD include edema, chest tightness, weight loss, and increased nocturnal awakenings.

A large multicenter trial suggests that dyspnea is a better predictor of mortality than spirometry in patients with COPD.[16] *Table 3* presents the Medical Research Council (MRC) dyspnea index, a validated clinical tool that physicians can use to quickly assess the severity of COPD.[17] The MRC dyspnea index has also been combined with body mass index (BMI), FEV_1, and exercise capacity (six-minute maximum walking distance) into the 10-point BODE index (*Table 4*).[18] The BODE index has been used to predict disease severity, risk of hospitalization, and all-cause mortality in patients with COPD.[3]

The differential diagnosis of COPD includes asthma, congestive heart failure, bronchiectasis, lung cancer, interstitial lung disease and pulmonary fibrosis, sarcoidosis, tuberculosis, and bronchopulmonary dysplasia. Asthma is the clinical disease that most often mimics COPD. Both are obstructive lung processes and can produce similar clinical symptoms. *Table 5* presents features useful in distinguishing asthma from COPD.[19,20]

PATIENT AND FAMILY HISTORY

An accurate patient history of tobacco use is another essential element of the clinical history in patients with suspected COPD. Cigarette smoking is best quantified using pack-years (the number of packs smoked per day multiplied by the number of years smoked). Similarly, a thorough occupational history is important when an occupational exposure is suspected. This includes a list of occupations; description of job-related activities; use of personal protective equipment, particularly respiratory protection; and length and extent of exposures. Material

Table 3. Medical Research Council Dyspnea Index

Grade	*Level of dyspnea*
1	Not bothered by dyspnea, except during strenuous activity
2	Shortness of breath when walking up a short hill
3	Walks more slowly than others because of breathlessness; stops to catch breath when walking at own pace
4	Stops to catch breath after walking 100 m (328 ft) on level ground
5	Too short of breath to leave the house; breathless with activities of daily living, such as dressing and undressing

Information from reference 17.

Table 4. BODE Index

	Points			
Variable	*0*	*1*	*2*	*3*
FEV_1 (percent of the predicted value)	≥ 65	50 to 64	36 to 49	≤ 35
Distance walked in six minutes (meters)	≥ 350	250 to 349	150 to 249	≤ 149
Grade on the MRC dyspnea scale (Table 3)	0 or 1	2	3	4 or 5
BMI (kg per m^2)	> 21	≤ 21	—	—

NOTE: *BODE index scores range from 0 to 10 points; higher scores indicate a greater risk of death.*

BMI = body mass index; BODE = BMI, airflow Obstruction, Dyspnea, Exercise capacity; FEV_1 = forced expiratory volume in one second; MRC = Medical Research Council.

Adapted with permission from Celli BR, Cote CG, Marin JM, et al. The body-mass index, airflow obstruction, dyspnea, and exercise capacity index in chronic obstructive pulmonary disease. N Engl J Med. *2004;350(10):1007.*

safety data sheets at occupation sites can be helpful to determine relative risks of occupational lung disease.

Family and occupational histories are also important when screening for COPD. $Alpha_1$-antitrypsin deficiency is a genetic anomaly of chromosome 14 that leads to premature hepatic and pulmonary disease. Patients with $alpha_1$-antitrypsin deficiency have early-onset COPD because of the increased tissue damage from neutrophil elastase. Alveolar damage leads to the loss of elastic recoil within the pulmonary parenchyma with subsequent airflow obstruction. Significant occupational hazards are associated with COPD in up to 15 percent of cases.[9]

It is estimated that 59,000 Americans have symptomatic COPD caused by $alpha_1$-antitrypsin deficiency.[21] Joint guidelines from the American Thoracic Society (ATS) and the European Respiratory Society (ERS) recommend screening for $alpha_1$-antitrypsin deficiency in symptomatic adults with persistent obstruction on pulmonary function testing. Screening is recommended in asymptomatic adults with a persistent obstruction on pulmonary function testing who also have a history of smoking or a significant history of occupational exposure.[22]

Significant occupational hazards are associated with COPD in up to 15 percent of cases.[9] Occupational hazards include exposure to dust, noxious gases, vapors, or fumes.[9] The industries with the highest exposure risk include those that work with rubber, plastics, leather, or textiles. Persons who work with utilities or general building construction are also at higher risk.[9] Laboratory studies have shown that coal, cadmium, and silica can cause alveolar destruction consistent with COPD.[9]

Table 5. Clinical Features Useful in Differentiating COPD from Asthma

Clinical feature	*COPD*	*Asthma*
Age	Older than 35 years	Any age
Cough	Persistent, productive	Intermittent, usually nonproductive
Smoking	Typical	Variable
Dyspnea	Progressive, persistent	Variable
Nocturnal symptoms	Breathlessness, late in disease	Coughing, wheezing; common
Family history	Less common	More common
Atopy	Less common	More common
Diurnal variation in symptoms	Less common	More common

COPD = chronic obstructive pulmonary disease.

Information from references 19 and 20.

Physical Examination

Physical examination findings are not sensitive for the initial diagnosis of COPD[23]; many patients have normal examination findings. In patients with abnormal findings, features of lung hyperinflation include a widened anteroposterior chest diameter, hyperresonance on percussion, and diminished breath sounds. Persistent pulmonary damage can lead to increased right-sided heart pressure causing right-sided heart failure (cor pulmonale). Signs of cor pulmonale on physical examination include an accentuated second heart sound, peripheral edema, jugular venous distension, and hepatomegaly. Signs of increased work of breathing include the use of accessory respiratory muscles, paradoxical abdominal movement, increased expiratory time, and pursed lip breathing; auscultatory wheezing is variable. Physical findings that are occasionally associated with COPD include cyanosis and cachexia.

COPD is associated with chronic weight

Table 6. COPD Staging Based on Spirometry Findings

Stage	*GOLD*[5]	*ATS/ERS*[30]	*BTS*[31]
0 (at risk)	—	Smokers with symptoms $FEV_1/FVC > 0.7$ $FEV_1 \geq 80$ percent of the predicted value	—
I (mild)	$FEV_1/FVC < 0.7$ $FEV_1 \geq 80$ percent of the predicted value	$FEV_1/FVC \leq 0.7$ $FEV_1 \geq 80$ percent of the predicted value	FEV_1 50 to 80 percent of the predicted value
II (moderate)	$FEV_1/FVC < 0.7$ FEV_1 50 to 79 percent of the predicted value	$FEV_1/FVC \leq 0.7$ FEV_1 50 to 80 percent of the predicted value	FEV_1 30 to 49 percent of the predicted value
III (severe)	$FEV_1/FVC < 0.7$ FEV_1 30 to 49 percent of the predicted value	$FEV_1/FVC \leq 0.7$ FEV_1 30 to 50 percent of the predicted value	$FEV_1 < 30$ percent of the predicted value
IV (very severe)	$FEV_1/FVC < 0.7$ $FEV_1 < 30$ percent of the predicted value or < 50 percent with chronic symptoms	$FEV_1/FVC \leq 0.7$ $FEV_1 < 30$ percent of the predicted value	—

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

Information from references 5, 30, and 31.

loss, which is an independent predictor of mortality.[24] It is important, therefore, to measure and monitor BMI in all patients with COPD. Clubbing is rarely associated with COPD and, when encountered, it should prompt a search for other causes such as cancer, pulmonary fibrosis, or bronchiectasis.[19]

Diagnostic Testing

SPIROMETRY

Suspected COPD should be confirmed using spirometry.[5,12,20,25] The National Heart, Lung, and Blood Institute recommends spirometry for all smokers 45 years or older, particularly those who present with shortness of breath, coughing, wheezing, or persistent sputum production.[26] Although spirometry is a simple, billable procedure that can be performed in the office,[27] it is underused by primary care physicians.[28,29]

The key spirometric features of COPD are FEV_1 and forced vital capacity (FVC). FEV_1 is the volume of air that a patient can expire in one second following a full inspiration. The FVC is the total maximum volume of air that a patient can exhale after a full inspiration. A postbronchodilator FEV_1/FVC ratio of less than 0.7 associated with an FEV_1 of less than 80 percent of the predicted value is diagnostic of airflow limitation and confirms COPD. A patient's FEV_1 relative to the predicted values in persons of similar age, sex, and height further characterizes the degree of airflow obstruction.

The ATS, ERS, Global Initiative for Chronic Obstructive Lung Disease, and British Thoracic Society have published guidelines for classifying COPD severity based on spirometry findings *(Table 6*[5,30,31]*)*. Disease severity is essential in determining the appropriate therapy for each patient. Spirometry can also be used to track disease progression over time. Peak expiratory flow rates are not helpful in diagnosing COPD because they can underestimate the level of airway obstruction.[19]

OTHER DIAGNOSTIC TESTS

Spirometry is the key test for diagnosing COPD; however, several additional tests are useful to rule out concomitant disease. Chest radiography should be performed to look for evidence of lung nodules, masses, or fibrotic change. Repeated or annual chest radiography and computed tomography to screen for lung cancer or to monitor the disease are not recommended. A complete blood count should be performed to exclude anemia or polycythemia. It is reasonable to perform electrocardiography and echocardiography in patients with signs of cor pulmonale to evaluate pulmonary circulatory pressures. Pulse oximetry at rest, with exertion, and during sleep should be performed to evaluate for hypoxemia and the need for supplemental oxygen.

The opinions and assertions contained herein are the private views of the authors and are not to be construed as official or as reflecting the views of the U.S. Navy Medical Department or the U.S. Navy Service at large.

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