THE JOURNAL OF FAMILY PRACTICE

Dean Gianakos, MD, and Rafal (Ralph) Kaczynski, MD
Lynchburg Family Medicine Residency, Lynchburg, VA
deangianakos@yahoo.com

Are your COPD patients benefiting from best practices?

Spirometry makes the diagnosis and determines therapy choices, yet it is vastly underused

Practice recommendations

- Perform spirometric testing on any patient who complains of difficulty breathing and has a history of smoking or risk factors for chronic obstructive pulmonary disease (COPD) (American College of Physicians grade: Strong recommendation, moderate-quality evidence)
- Use inhaled bronchodilators and oral glucocorticosteroids for COPD exacerbations (Global Initiative for Chronic Obstructive Lung Disease [GOLD] Evidence **A**)
- Use antibiotics for COPD exacerbations (GOLD Evidence **B**)
- Use long-acting beta-agonists, long-acting anticholinergics, or inhaled steroids for chronic, stable COPD (American College of Physicians grade: Strong recommendation, high-quality evidence)
- Smoking cessation is the most effective way to decrease the risk of COPD progression (GOLD Evidence **A**)

GOLD Evidence categories

A Randomized controlled trials (RCTs); rich body of data
B RCTs; limited body of data
C Nonrandomized trials; observational studies
D Panel consensus judgment

A new patient comes into your office and tells you he experiences labored breathing on exertion, smokes a pack of cigarettes a day, and has a smoker's cough.

- Would you perform spirometry to gauge airway obstruction?
- How do you think your decision would compare with those of your colleagues?

In this article, we put your answer into context by revealing just how underutilized spirometry is.

We also use a progressive case example to illustrate evidence-based recommendations and management tips for chronic obstructive pulmonary disease (COPD) and address often overlooked gaps in care.

Many of the recommendations in this article come from the Global Initiative for Chronic Obstructive Lung Disease (GOLD), published in the *American Journal of Respiratory and Critical Care Medicine*[1] and updated online at www.goldcopd.org. (This initiative, begun in 1998, provides specific, evidence-based guidelines on the prevention, assessment, and management of COPD patients.) We also refer to newly published American College of Physicians (ACP) evidence-based guidelines for managing chronic, stable COPD.[2,3]

IN THIS ARTICLE

CASE Shortness of breath, smoker's cough

Mr. Jones, a 57-year-old patient in our practice, says that for the past 3 months he has increasingly experienced shortness of breath when walking up a flight of stairs. He has smoked cigarettes for many years and also acknowledges having a smoker's cough. He brings up clear phlegm on most days.

Dyspnea is the most common symptom reported by patients with COPD. In a study of 2678 patients, the first and most troublesome symptom noted was dyspnea (71%), followed by cough (19%).[4] Patients typically say their dyspnea has worsened over time. It tends to occur daily, particularly with exercise. Cough may be intermittent and nonproductive.

Consider the diagnosis whenever a patient with dyspnea has a risk factor for COPD, such as smoking (~80% of cases); extended second-hand smoke exposure; contact with occupational dust, home cooking and heating fuels, or other potentially toxic chemicals; or has a history of recurrent lung infections.[5] With patients in their 30s or 40s exhibiting signs and symptoms suggestive of COPD, consider a work-up for alpha-1 antitrypsin deficiency. (See "Does your patient have alpha-1 antitrypsin deficiency?" on page 535.)

Physical examination has limited usefulness. It exhibits poor sensitivity for detecting mild-to-moderate COPD, unless wheezing is present. Wheezing in smokers (more than 40 pack-years) has a positive likelihood ratio of 8.3 for obstructive airway disease.[6]

Physical diagnosis is easier with more severe disease, especially if patients show classic signs of COPD, such as pursed-lip breathing, decreased breath sounds, and prolonged expiratory wheezes.

Spirometry is key, and underused. Demonstrating airflow obstruction on spirometry is essential to a COPD diagnosis. An FEV_1/FVC ratio <0.70 or FEV_1 <80% in patients who have received a test-bronchodilator confirms airflow obstruction.

Amazingly, a COPD diagnosis is assigned to less than half of the estimated 24 million patients with airflow obstruction in United States,[7] despite the fact that COPD is the 4th leading cause of death, and the 12th leading cause of morbidity.[1] Most of those who are identified have advanced disease.[8] This dramatic underdiagnosis is attributable to the underuse of office spirometry as a diagnostic tool.[9]

A Canadian study revealed that only 21% of physicians ordered spirometry when managing a middle-aged smoker with cough.[10,11] Another study showed that only 22% of North American physicians would order spirometry for a smoker with cough.[10,12] Only a third of patients had undergone spirometry within 2 years of a new diagnosis of COPD. The lowest frequency of testing was among elderly patients, especially among those older than 75 years.[10] (Caveat: as patients age, FEV_1 naturally declines, making it easy to overdiagnose airflow obstruction in elderly patients.[8,13])

The above data regarding underuse of spirometry apply to symptomatic patients. A recent US Preventive Services Task Force analysis found that screening asymptomatic smokers does not improve health outcomes; the number needed to test with spirometry would be in the "hundreds" to defer a single exacerbation.[9] (ACP grade: strong recommendation, moderate-quality evidence.)

Reserve chest radiographs and CT scans to rule out other disorders. Patients with COPD usually have elements of both chronic bronchitis (productive cough for 3 months in 2 consecutive years) and emphysema (defined anatomically as abnormal enlargement of airways distal to terminal bronchioles and destruction of alveolar walls). Radiographic tests may reveal the telltale signs of emphysema (flattened diaphragms, blebs, and bullous changes), but they are not necessary to make the diagnosis. They may be used, however, to exclude other causes of dyspnea, including congestive heart failure, pulmo-

FAST TRACK

The dramatic underdiagnosis of COPD is attributable to underuse of office spirometry

THE JOURNAL OF FAMILY PRACTICE

Managing stable COPD: ACP recommendations at a glance*

Recommendation 1 With patients who have respiratory symptoms, particularly dyspnea, perform spirometry to diagnose airflow obstruction. Spirometry should not be used to screen for airflow obstruction in asymptomatic individuals. (**Grade:** strong recommendation, moderate-quality evidence.)

Recommendation 2 Reserve treatment for patients who have respiratory symptoms and an FEV_1 <60% predicted, as documented by spirometry. (**Grade:** strong recommendation, moderate-quality evidence.)

Recommendation 3 Prescribe 1 of the following maintenance monotherapies for symptomatic patients with COPD and an FEV_1 <60% predicted: long-acting inhaled beta-agonists, long-acting inhaled anticholinergics, or inhaled corticosteroids. (**Grade:** strong recommendation, high-quality evidence.)

Recommendation 4 You may want to consider combination inhaled therapies for symptomatic patients with COPD and an FEV_1 <60% predicted. (**Grade:** weak recommendation, moderate-quality evidence.)

Recommendation 5 Prescribe oxygen therapy for patients with COPD and resting hypoxemia (PaO_2 ≤55 mm Hg). (**Grade:** strong recommendation, moderate-quality evidence.)

Recommendation 6 Consider prescribing pulmonary rehabilitation for symptomatic individuals with COPD who have an FEV_1 <50% predicted. (**Grade:** weak recommendation, moderate-quality evidence.)

*Modified from Qaseem A et al. Diagnosis and management of stable chronic obstructive pulmonary disease: a clinical practice guideline from the American College of Physicians. *Ann Intern Med.* 2007;147:633-638.

nary emboli, and interstitial lung disease (**TABLE**).

CASE Spirometry reveals airflow obstruction, FEV_1 <50%

Mr. Jones underwent spirometry, which revealed airflow obstruction and an FEV_1 <50%. We gave him a short-acting beta-agonist to be used as needed. Two weeks later, he returned to the office with increasing cough and purulent sputum production, as well as worsening dyspnea.

The patient's condition is consistent with an acute exacerbation of baseline COPD symptoms. Worsening dyspnea, cough, and sputum production—sometimes with purulence—are often accompanied by fever, fatigue, and anorexia.[7]

Match antibiotic therapy to sputum culture results or disease severity. Exacerbations are usually triggered by infection. Although an offending organism cannot be identified in one third of cases, common bacterial pathogens include *Hemophilus influenza*, *Streptococcus pneumoniae*, and *Moraxella catarrhalis*. Antibiotics have been shown to decrease mortality in patients with COPD exacerbations[1,5,7,14,15] (GOLD Evidence B). For mild-to-moderate exacerbations, older antibiotics such as trimethoprim/sulfamethoxazole or doxycycline are often appropriate. For more severe exacerbations, and for pa-

tients with chronic, comorbid conditions such as diabetes mellitus, a second- or third-generation cephalosporin or fluoroquinolone may be preferable.

Use steroids and beta-agonists. Oral steroids are also effective in treating exacerbations (GOLD Evidence A), although the dose of steroids required has not been adequately studied. Prednisone, 40 mg/d for 7 to 10 days, is reasonable and safe.[1] Also prescribe an inhaled short-acting beta-agonist for symptom control (GOLD Evidence A).[1]

CASE Doxycycline 100 mg bid, and prednisone 40 mg/d for 7 days

Mr. Jones returned to the office 2 weeks after the acute exacerbation, feeling much better after receiving doxycycline 100 mg bid and prednisone 40 mg/d for 7 days. He was no longer coughing up purulent sputum, but he still felt short of breath walking to his mailbox and while doing household chores. He wondered what else could be done to improve his quality of life.

The airflow obstruction associated with COPD, unlike that of asthma, is irreversible and varies little,[16] and its progression is persistent. That is why prevention is an important goal for physicians and their patients. However, treatment can lessen the frequency of exacerbations and severity of symptoms, particularly dyspnea on exertion.

In our initial assessment of the patient, his FEV_1 was <50%. There was no need to repeat spirometry, as the evidence does not support ongoing spirometric evaluation.[2,3] Symptomatic patients with significant airflow obstruction (FEV_1 <60% predicted) are the ones most likely to benefit from therapy (ACP grade: strong recommendation, moderate-quality evidence).[2,3] Conversely, there is little evidence to justify treating asymptomatic patients who have airflow obstruction.

Monotherapy with long-acting inhaled beta-agonists, inhaled corticosteroids, or long-acting inhaled anticholinergics has been shown to reduce exacerbations and is preferable to short-acting, inhaled beta-agonists or short-acting anticholinergics (ACP grade: strong recommendation, high-quality evidence).[2,3] At this time, evidence is insufficient to support the use of combined therapies—eg, inhaled steroids plus long-acting beta-agonists.[2,3]

For patients with a PaO_2 ≤55 mm Hg, survival is improved by using supplemental oxygen therapy for 15 or more hours a day. (ACP grade: strong recommendation, moderate quality evidence).[2,3]

Finally, for symptomatic patients with an FEV_1 <50%, pulmonary rehabilitation may reduce hospitalizations and increase exercise capacity (ACP grade: weak recommendation, moderate-quality evidence).[2,3]

For Mr. Jones, we prescribed 1 inhalation daily of the long-acting anticholinergic inhaler, tiotropium.

Smoking cessation critical

COPD progresses with aging and with continued smoking, and smoking cessation is critical to any management strategy.

CONTINUED

Does your patient have alpha-1 antitrypsin deficiency?

Alpha-1 antitrypsin deficiency is an autosomal recessive disorder that causes COPD and liver cirrhosis.[17] Alpha-1 antitrypsin protects the lungs from proteases released from inflammatory processes such as pneumonia and from inhaled toxic particles. When this glycoprotein is absent, proteases destroy airways and alveoli. Consider the diagnosis with younger patients. The disease is easily confused with asthma or smoking-induced COPD. It predominantly affects the lower lobes. Diagnosis is made by testing blood levels for the enzyme or genetic analysis.

Treatment is the same as for other causes of COPD. Although no evidence-based recommendations are available at this time, replacement of alpha-1 antitrypsin is indicated for certain patients. Smoking cessation is critical.

FAST TRACK

Alpha-1 antitrypsin deficiency is easily confused with asthma or smoking-induced COPD

TABLE

Suspect COPD? Rule out these disorders

DISORDER	NOTABLE CHARACTERISTICS
Asthma	Usually begins in childhood. Can be associated with cough only. Airflow obstruction is usually reversible with bronchodilator (may coexist with COPD)
Cystic fibrosis	Symptoms usually begin in early childhood. Associated with sinus disease, GI disturbances, and infertility. Bronchiectasis noted on chest x-ray. Order sweat chloride test if suspected. Genetic testing is also available
Interstitial lung disease	Interstitial pattern on chest x-ray and thin-cut CT scan of lungs
Pneumonia	Fever, chills, cough, and infiltrate on chest x-ray
Congestive heart failure	Orthopnea, paroxysmal nocturnal dyspnea, and characteristic chest x-ray findings
Pulmonary embolism	Breathing difficulty and chest pain usually of sudden onset. CT angiography is diagnostic
Anxiety	Hyperventilation, panic attacks, increased stress

FAST TRACK

For patients with PaO_2 ≤55 mm Hg, survival is improved by using oxygen therapy for ≥15 hours a day

CASE Tiotropium, 1 inhalation daily, and a smoking cessation plan

We referred Mr. Jones to an outpatient smoking cessation program and gave him American Academy of Family Physicians patient education materials to review. His exercise tolerance improved with 1 inhalation daily of the long-acting anticholinergic inhaler, tiotropium, and he is making progress in his efforts to quit smoking. ■

Correspondence

Dean Gianakos, MD, Lynchburg Family Medicine Residency, 2097 Langhorne Road, Lynchburg, VA 24501; deangianakos@yahoo.com

Disclosure

The authors reported no potential conflict of interest relevant to this article.

References

1. Rabe KF, Hurd S, Anzueto A, et al. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease (GOLD). *Am J Respir Crit Care Med.* 2007;176:532-555.
2. Qaseem A, Snow V, Shekelle P, et al. Diagnosis and management of stable chronic obstructive pulmonary disease: a clinical practice guideline from the American College of Physicians. *Ann Intern Med.* 2007;147:633-638.
3. Wilt TJ, Niewoehner D, MacDonald R, et al. Management of stable chronic obstructive pulmonary disease: a systematic review for a clinical practice guideline. *Ann Intern Med.* 2007;147:639-653.
4. Kesten S, Menjoge S. Patient-reported symptoms of chronic obstructive pulmonary disease in clinical trials. *Chest.* 2005;128(4):249S.
5. Pauwels RA, Buist AS, Ma P, et al. GOLD Scientific Committee. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease. *Respir Care.* 2001;46:798-825.
6. Straus SE, McAlister FA, Sackett DL, et al. The accuracy of patient history, wheezing, and laryngeal measurements in diagnosing obstructive airway disease. *JAMA.* 2000;283:1853-1857.
7. Wise RA, Tashkin DP. Optimizing treatment of chronic obstructive pulmonary disease: an assessment of current therapies. *Am J Med* 2007;120(8A):S4-S13.
8. Lin K, Watkins B, Johnson T, et al. Screening for chronic obstructive pulmonary disease using spirometry: summary of the evidence for the U.S. Preventive Services Task Force. *Ann Intern Med.* 2008;148:535-543.
9. Sundblad BM, Larsson K, Nathell L. Low awareness of COPD among physicians. *Clin Respir J.* 2007;(1):11-16.
10. Han M, Kim MG, Mardon R, et al. Spirometry utilization for COPD: how do we measure up? *Chest.* 2007;132:403-409.
11. Kesten S, Chapman K. Physician perceptions and management of COPD. *Chest.* 1993;104:254-258.
12. Chapman K, Tashkin D, Pye D. Gender bias in the diagnosis of COPD. *Chest.* 2001;119:1691-1695.
13. Nazir SA, Al-Hamed MM, Erbland ML. Chronic obstructive pulmonary disease in the older patient. *Clin Chest Med.* 2007;28:703-715.
14. Ram FS, Rodriguez-Roisin R, Granados-Navarrete A, et al. Antibiotics for exacerbations of chronic obstructive pulmonary disease. *Cochrane Database Syst Rev.* 2006;(2):CD004403.
15. Littner M. In the clinic. Chronic obstructive pulmonary disease. *Ann Intern Med.* 2008;148(5):ITC3-1-ITC3-16.
16. Dewar M, Curry W. Chronic obstructive pulmonary disease: diagnostic considerations. *Am Fam Physician.* 2006;73:669-678.
17. Kohnlein T, Welte T. Alpha-1 antitrypsin deficiency: pathogenesis, clinical presentation, diagnosis, and treatment. *Am J Med.* 2008;121:3-9.