

# Primary care of chronic dyspnea in adults

Abstract: Chronic dyspnea is the presence of shortness of breath lasting for a minimum of 4 weeks. It is a common complaint associated with many of the cardiopulmonary diseases seen in primary care. This article provides a systematic, evidence-based, and cost-effective approach to the evaluation of this complex symptom.

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yspnea is a common complaint that is defined as "a subjective experience of breathing discomfort that consists of qualitatively distinct sensations that vary in intensity."<sup>1</sup> Dyspnea is a multifactorial, complex symptom that is not well-understood. Previously, the symptom of dyspnea was evaluated primarily from the physiologic domain with less consideration of the emotional or psychological impact the symptom had on the individual. As dyspnea occurs, the patient often becomes anxious, leading to an increased sensation of dyspnea. Providers often tell dyspneic patients to calm down and to control their breathing. However, some patients have reported increased anxiety and shortness of breath in this situation.<sup>2</sup>

A patient's complaint of dyspnea may indicate a broad spectrum of causes from simple physical deconditioning to more serious and life-threatening conditions. As healthcare providers, it is imperative that nurse practitioners (NPs) be aware of the complexity of the evaluation regarding the symptom of dyspnea.

Chronic dyspnea is the presence of shortness of breath for at least 4 weeks.<sup>3</sup> Dyspnea may be reported by the patient as mild to severe, although the perceived level of dyspnea may not correlate with the severity of the underlying cause.<sup>2</sup> According to recent research, some of the dyspnea sensation descriptors given by patients may provide clues to causation. Patients who complain of air hunger may have heart failure (HF); chest tightness is highly suggestive of bronchoconstriction; and the complaint of having a "hard time" breathing may be related to chronic obstructive pulmonary disease (COPD), interstitial lung disease (ILD), or physical deconditioning.<sup>4-6</sup>

Understanding the pathophysiology of dyspnea continues to evolve. There are two, primary general categories of dyspnea causation: pulmonary-related and cardiovascularrelated.<sup>4,6</sup> Those that are cardiovascular related also include anemia and physical deconditioning.<sup>6</sup> Dyspnea is recognized as having neurosensory processing similar to nociceptive pain processing.<sup>4</sup> This understanding strongly supports the difference in patient perception of dyspnea with the same severity of underlying physiologic cause. For example, two patients with the same degree of COPD may express very different perceptions of dyspnea, though clinical and physiologic data are similar. The majority of patients with chronic dyspnea are eventually diagnosed with cardiovascular or respiratory disorders. Less common causes include psychiatric, gastrointestinal, and neuromuscular disorders.

Keywords: adults, cardiopulmonary disease, dyspnea, primary care, shortness of breath

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The evaluation of chronic dyspnea begins with a thorough history and physical.

# History

The patient's history is crucial in the evaluation of dyspnea. Historical information will likely reveal variation in the subjective description of dyspnea as well as the patient's own perception of its causation. Patients should be asked about the timing, setting, and any aggravating or alleviating factors related to their dyspnea. They should also be asked if the dyspnea occurs at rest or with exertion. The effect of shortness of breath on activities of daily living should be quantified. The ability or inability to ambulate, climb stairs, and perform specific activities of daily living should be documented. Providers should determine how the dyspnea impacts sleep and if the patient experiences orthopnea or paroxysmal nocturnal dyspnea. A detailed history of chronic dyspnea can provide clues to guide exam and diagnostic evaluation.

A thorough social history should include home and occupational environments. Personal tobacco use, exposure to tobacco smoke, and other irritant substances-both at home and at work-should be questioned. Irritants include chemical fumes, molds, allergens, and other substances that may cause or aggravate the patient's dyspnea.<sup>6</sup> A full review of the patient's current medications, including supplements and herbal preparations, should be conducted. While the medications that the patient is taking rarely cause dyspnea, there are a few that should be specifically considered. If a patient has recently been prescribed a nonselective beta-blocker and has worsening dyspnea, it is possible that the patient has undiagnosed asthma and is having medication-induced bronchospasm, which is triggering the shortness of breath. Long-term use of nitrofurantoin for suppression of recurrent urinary tract infections has been well documented for its association with ILD.7 Chemotherapeutic drugs, amiodarone, hydrochlorothiazide, and aspirin have also been implicated in causing shortness of breath.7

The review of systems should thoroughly evaluate for potential symptoms related to dyspnea (see *Targeted review of systems for chronic dyspnea*).<sup>8</sup>

# Physical exam

A comprehensive physical exam should be performed. Vital signs with height, weight, calculated body mass index, and pulse oximetry at rest should be measured. Normal pulse oximetry will not rule out a pulmonary cause. The general appearance of the patient and any presence of distress should be noted. Skin color should be evaluated for pallor or cyanosis, and nails should be assessed for clubbing. The skin should also be assessed for eczema, which may be present in the patient with allergies and asthma. A thorough exam of the eyes, ears, nose, throat, and sinuses to evaluate for allergies, postnasal drip, and chronic sinusitis should be performed. The neck should be evaluated for the presence of thyromegaly, and jugular vein distension should be estimated. The thorax should be evaluated for the presence of spinal, rib cage, or sternal deformities that could cause restriction of the chest cavity, and therefore, chronic shortness of breath.

The ease of respirations, use of accessory muscles, symmetry of chest excursion, and the respiratory rate and depth should be noted while at rest. The lungs should be percussed for areas of dullness (consolidation or atelectasis) or diffuse hyperresonance (air-trapping). Diminished breath sounds or the presence of crackles or wheezes should be noted. Cardiac rate, rhythm, location of the point of maximal impulse (apical impulse), and presence of extra heart sounds and murmurs should be documented. The abdomen should be examined for hepatomegaly, hepatojugular reflex, and presence of ascites.<sup>5</sup> Peripheral extremities should be evaluated for presence of edema and for coolness.<sup>5,9</sup> The presence of any of these abnormal physical exam findings should help identify likely causes of dyspnea and direct further evaluation of the patient.

### Common causes of chronic dyspnea

The majority of patients with dyspnea will be diagnosed with cardiac or respiratory disorders, including asthma, COPD, ILD, chronic pneumonia, HF, atrial fibrillation, and myocardial ischemia.<sup>5,10</sup>

Patients with asthma may complain of chest tightness and shortness of breath with or without wheezing or with cough only. However, wheezing and/or cough are not specific to respiratory conditions and may also be present in the patient with HF or other conditions. Patients with asthma may be able to identify a trigger, such as weather changes or a particular respiratory irritant that precipitates the symptoms. Patients with asthma often have worsening of symptoms at night and symptoms that interfere with sleep.

Patients with COPD will typically present with dyspnea and a productive cough. Most patients with COPD are smokers<sup>11</sup>; however, there is an increased worldwide prevalence of nonsmokers who have COPD due to significant exposure to second-hand smoke, pollution, and other respiratory irritants. ILD is the last common pulmonary cause of dyspnea. ILD is not a single disorder but rather a group of over 100 disorders that have progressive dyspnea as a symptom.<sup>12</sup> ILD may be due to several different connective tissue disorders, exposure to various respiratory

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Major body systems	Symptoms and pertinent history findings
General	• Fatigue, fever, rapid weight gain
Head, eyes, ears, nose, and throat	<ul> <li>Rhinorrhea, nasal congestion, nasal polyps, sore throat, hoarseness</li> <li>History of recurrent colds</li> </ul>
Respiratory	<ul> <li>Cough, sputum (character), hemoptysis, wheezing</li> <li>History of asthma, emphysema, or pneumonia</li> <li>Date of last chest X-ray</li> </ul>
Cardiovascular	<ul> <li>Chest pain, palpitations, orthopnea, paroxysmal nocturnal dyspnea, edema</li> <li>History of hypertension, angina, or myocardial infarction</li> </ul>
Gastrointestinal	<ul> <li>Heartburn, difficulty swallowing, abdominal discomfort, constipation, diarrhea</li> </ul>
Peripheral vascular	<ul><li>Varicose veins</li><li>History of deep vein thrombosis</li></ul>
Neuromuscular	Decreased sensation, muscle weakness
Hematologic	<ul><li>Bruises easily, bleeding</li><li>History of anemia</li></ul>
Endocrine	<ul><li>Cold or heat intolerance</li><li>History of thyroid problems</li></ul>
Psychiatric	Anxiety, depression, panic episodes

irritants, medications, or infections. There are other less common respiratory causes of chronic dyspnea, such as postnasal drainage, chronic pneumonia, or chronic pulmonary embolism. Chronic postnasal drainage rarely presents with the chief complaint of chronic dyspnea and is generally determined early in the physical exam.<sup>13</sup>

Cardiac disease presenting with chronic dyspnea is most commonly due to either HF, myocardial ischemia, or cardiac dysrhythmia, typically atrial fibrillation. Patients with HF exacerbation may present with complaints of suffocating or worsening dyspnea with activity.<sup>5</sup> They may also present with cough or wheezing. Patients with left-sided HF (systolic or diastolic dysfunction) may have fatigue, cough, and dyspnea that interferes with sleep due to orthopnea or episodes of paroxysmal nocturnal dyspnea. Patients with right-sided HF may present with complaints of increased weight gain, frequent urination, and dyspnea that is due to abdominal distension from liver enlargement and possible ascites. On exam, elevated jugular venous pressure, peripheral edema, and tenderness in the right upper abdomen associated with hepatomegaly may be found. A positive hepatojugular reflex (distension of jugular veins with pressure over the liver) may be elicited.

## Diagnostic studies

Evaluation for chronic dyspnea begins with basic lab studies. Patients should have (at minimum) a complete blood cell count to evaluate for anemia and a thyroid-stimulating hormone (TSH) to evaluate for hypothyroidism. A comprehensive metabolic panel should be part of the initial labs to evaluate both hepatic and renal function. Additional initial testing should be based on the provider's suspected diagnosis, although a plain chest film is recommended for most patients even with a normal cardiopulmonary exam.<sup>3,13</sup> A plain chest film may reveal the presence of hyperinflation, consolidation (tumor or pneumonia), cardiomegaly, or pulmonary congestion.

Spirometry is indicated if a pulmonary cause is suspected. The postbronchodilator testing with at least partial reversibility of airflow limitation (improvement of at least 12% of the forced expiratory volume in 1 second [FEV1]) can confirm the diagnosis of asthma if obstruction is evident.<sup>14</sup> Persistent airflow limitation, defined as the ratio of FEV1/FVC less than 0.70, is suggestive of COPD.<sup>15</sup> Both asthma and COPD demonstrate obstructive patterns on spirometry. ILD and other disorders should be considered if the total lung capacity is decreased, which is suggestive of a restrictive disorder.<sup>16</sup> Patients with restrictive disorders are typically referred to a pulmonologist for management.

An ECG should be performed if the cause is thought to be of cardiac origin, and a brain-type natriuretic peptide (BNP) should be drawn. Elevations of BNP greater than 100 pg/mL are suggestive of HF.<sup>9</sup> These initial diagnostic tests are evidence-based, readily accessible, with relatively low-cost and low-risk to the patient.

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Pratter et al. developed an algorithm for the evaluation of chronic dyspnea.<sup>13</sup> The study was conducted with 123 patients with undiagnosed dyspnea lasting at least 8 weeks. The majority of the patients were able to be diagnosed using a three-tiered approach, which was efficient and costeffective. All patients had a plain chest film, pulmonary function testing, and blood work. Labs included a basic chemistry panel, complete blood cell count, TSH, and BNP. About a third of the patients were able to be diagnosed with these tests. Those undiagnosed proceeded to have cardiopulmonary exercise testing (CPET), which allowed for the diagnosis of another third of the patients. The final third group of patients proceeded to organ-specific and often invasive testing. All but one patient was able to be diagnosed using this approach.<sup>13</sup>

If cardiopulmonary disorders are still suspected but not clearly diagnosed with the initial evaluation and testing, the primary care provider should refer the patient for CPET and possibly an echocardiogram.<sup>6,13,16</sup> CPET can be useful in multiple situations when the diagnosis of chronic dyspnea remains unclear. The test can help

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distinguish between cardiac or pulmonary causation of dyspnea in the patient who has disorders affecting both systems.<sup>17</sup> For example, the CPET can help determine which disorder is causing the dyspnea in the patient with both HF and COPD. The test can also help determine if physical deconditioning is the cause of the shortness of breath.<sup>17</sup> Less common causes of chronic dyspnea such as psychogenic disorders, gastroesophageal reflux, and neuromuscular disorders should be considered.

# Treatment

Appropriate treatment can be initiated once the underlying cause of the chronic dyspnea has been determined. The majority of conditions that cause chronic dyspnea can be safely and effectively managed by primary care providers.

Asthma. Once the diagnosis of asthma is confirmed by spirometry, the patient's asthma should be classified based on the criteria from the National Asthma Education and Prevention Program (NAEPP) guidelines.<sup>14</sup> Once the severity of asthma is classified, treatment is then initiated using the step approach outlined by NAEPP.<sup>14</sup> The initial approach is to gain control of the symptoms and improve the quality of life while decreasing the risk of morbidity and mortality. The majority of patients will require inhaled corticosteroids and other adjunctive medications to manage their asthma. Once initial treatment is started, the focus of care becomes controlling asthma. As asthma control is documented over time, the treatment is "stepped down" to lower doses (or fewer medications) as long as symptom control is maintained.<sup>14</sup>

**COPD.** The diagnosis of COPD is supported by spirometry (postbronchodilator FEV1/FVC ratio of less than 70%) in conjunction with evaluation of symptoms and risk. The treatment is guided by the Global Initiative for Chronic Obstructive Lung Disease (GOLD) updated 2014 guide-lines.<sup>15</sup> The most important treatment for a patient with COPD who smokes is cessation. Pharmacologic treatment of COPD is directed toward symptom improvement, as there is no current evidence that long-term physiologic outcomes are affected by medications. The main treatment is through various inhaled bronchodilators: short-acting or long-acting beta<sub>2</sub>-agonists and anticholinergic drugs. The beta<sub>2</sub>-agonists inhalers, such as albuterol, may be given as needed or on a

regular basis. The newest class of drug is the phosphodiesterase-4 inhibitor. Roflumilast is an oral medication that has been shown to improve FEV1 in patients using long-acting bronchodilator therapy.<sup>15</sup> The drug has also been shown to decrease COPD exacerbations in patients with severe diseases. Patients

with COPD over the age of 65 or those who are younger with other chronic comorbid conditions should receive the pneumococcal vaccine. All patients with COPD should receive an annual influenza vaccine.<sup>15</sup> Oxygen therapy may be necessary in some patients, and oxygen saturation should be measured twice during a 3-week period. The patient should receive home oxygen therapy if the oxygen saturation is less than 88% on both measures.<sup>15</sup> Pulmonary rehabilitation for a minimum of 6 weeks has also shown to be beneficial for patients with COPD.<sup>4</sup>

HF. According to the 2013 American College of Cardiology Foundation/American Heart Association (ACCF/AHA) guidelines, the term "congestive heart failure" should not be used, as it is an inaccurate descriptor for many with HF diagnosis.<sup>9</sup> Therefore, the term "heart failure" is preferred. The initial workup should include additional testing of urinalysis, serum lipids, and a chemistry panel that includes renal and liver functions if the patient is found to have HF as the cause of chronic dyspnea. All patients, regardless of the class of HF they have, should be initially treated with an angiotensin-converting enzyme-inhibitor or an angiotensin II receptor blocker plus one of three specifically recommended beta-blockers (bisoprolol, carvedilol, sustained release metoprolol succinate) that have been demonstrated to reduce mortality in stable HF patients.<sup>9</sup> This combination of drugs should be used unless contraindicated, even in Black patients and those with comorbid diabetes. Further drug choice depends on the presence of volume overload, the specific New York Heart Association HF class, renal function, and if the patient is Black. If the patient has hypertension or hyperlipidemia, in addition to the new diagnosis of HF, the patient should have therapy maximized for those comorbid conditions. As in all conditions, if the patient uses tobacco, the patient should be counseled and assisted with smoking cessation. The patient should also be referred to a cardiologist for further evaluation and management.

Atrial fibrillation. The presence or absence of underlying heart disease must be determined to guide treatment of the AF for the patient with new onset atrial fibrillation (AF) as the cause of chronic dyspnea.<sup>18</sup> There is an unknown prevalence of "lone" AF (idiopathic) seen in young patients without identifiable causation from cardiopulmonary disorders.<sup>19</sup> The risk of thromboembolic events in this group is unknown. In the latest ACCF/AHA guidelines for the treatment of AF, it is recommended that antithrombotic therapy be given for all patients unless contraindicated. Patients with "lone" AF are the exception and should not receive antithrombotic medications.<sup>18</sup> Eighty-one to 325 mg of daily aspirin is recommended for patients identified as low-risk or those who have contraindications to oral anticoagulant therapy.<sup>18</sup> Oral anticoagulation is required for patients with comorbid conditions or artificial heart valves. As of 2011, it is no longer recommended to attempt to control the resting heart rate to 80 beats/minute in patients with asymptomatic, stable AF.18 Patients with newly diagnosed AF should be referred to a cardiologist for further evaluation and management.

**GERD**. Gastroesophageal reflux disease rarely presents without the patient reporting some of the typical symptoms associated with the disease, such as heartburn or regurgitation.<sup>20</sup> GERD may less commonly present with "extraesophageal" symptoms, such as noncardiac chest pain, chronic cough, or hoarseness.<sup>20</sup> Dyspnea as a presenting symptom is not included in the current guidelines for diagnosis and treatment. However, a trial of a proton-pump inhibitor (PPI) would be suggested if the patient is suspected to have GERD as a cause of chronic dyspnea and complains of typical reflux symptoms. If the patient does not improve after the appropriate trial of PPI used for GERD, the patient should be referred for further workup.<sup>20</sup>

**Psychogenic causes.** Psychogenic causes were found to be the most common noncardiopulmonary cause of the complaint in various studies of patients with chronic

dyspnea.<sup>3,5,13</sup> Generalized anxiety disorder, panic disorder, and depression have been identified as potential diagnoses with chronic dyspnea. Patients with generalized anxiety disorder may develop hyperventilation and complain of dyspnea. These patients have often had extensive diagnostic workups in the ED to rule out causes for acute episodes of dyspnea. However, the acute problem of dyspnea can develop into a chronic condition for patients without diagnosis of the underlying cause. Patients with anxiety that presents as chronic dyspnea generally complain of dyspnea at rest that improves with distraction or exertion.<sup>10</sup> In addition, perioral or facial paresthesia is an associated symptom with hyperventilation that may be due to anxiety. Anxiety is not uncommon in the patient with cardiopulmonary disorders, so it should not be diagnosed until other common causes are ruled out. Anxiety may also occur as a comorbid condition in patients with depression, which is recognized as common in patients with chronic health disorders. Primary care screening tools are available for both anxiety and depression.<sup>21,22</sup>

**Physical deconditioning.** Patients with normal cardiopulmonary function may experience dyspnea secondary to deconditioning from maintaining a relative sedentary state.<sup>6</sup> Patients with chronic cardiopulmonary diseases may choose to become less active in an attempt to conserve energy and reduce fatigue. Unfortunately, the long-term effect leads to decreased physical ability and increased dyspnea with minimal exertion due to deconditioning.<sup>6</sup>

# Moving forward

As treatment of chronic disease improves and the population ages, chronic dyspnea will likely become an increasingly common symptom seen in primary care. Currently, there is no single diagnostic test available to fully evaluate the symptom of dyspnea. Therefore, the provider must thoroughly evaluate the patient through history and physical exam and must begin a diagnostic workup. The first round of testing should include basic lab studies, ECG, and a plain chest film. Spirometry is often available in many primary care offices and may be part of the initial workup if a high index of suspicion exists for pulmonary disease. If the cause of dyspnea remains unclear after the initial workup, the patient should begin the next round of diagnostic testing or receive a referral for further evaluation.<sup>23</sup> The majority of patients will be able to be quickly assessed, diagnosed, and receive prompt treatment if this type of approach is followed.

# REFERENCES

- American Thoracic Society. Dyspnea. Mechanisms, assessment, and management: a consensus statement. Am J Respir Crit Care Med. 1999;159(1):321-340.
- 2. McCarley C. A model of chronic dyspnea. *Image J Nurs Sch.* 1999;31(3): 231-236.

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- 3. Peters SP. When the chief complaint is (or should be) dyspnea in adults. *J Allergy Clin Immunol Pract.* 2013;1(2):129-136.
- 4. Parshall MB, Schwartzstein RM, Adams L, et al. An official American Thoracic Society statement: update on the mechanisms, assessment, and management of dyspnea. *Am J Respir Crit Care Med.* 2012;185(4):435-452.
- 5. Wahls SA. Causes and evaluation of chronic dyspnea. *Am Fam Physician*. 2012;86(2):173-182.
- 6. Schwartzstein RM. Approach to patient with dyspnea. In: King TE Jr, ed. Up-ToDate. Waltham, MA.
- Fagan NL, Foral PA, Malesker MA, Morrow LE. Therapeutic update on druginduced pulmonary disorders. US Pharm. 2011;36(7):HS3-HS8.
- 8. Bickley LS. Bates' Guide to Physical Examination and History Taking. 11th ed. Philadelphia: LWW; 2013:10-13.
- Yancy CW, Jessup M, Bozkurt B, et al. 2013 ACCF/AHA guideline for the management of heart failure: executive summary: a report of the American College of Cardiology Foundation/American Heart Association Task Force on practice guidelines. *Circulation*. 2013;128(16):1810-1852.
- 10. Sarkar S, Amelung PJ. Evaluation of the dyspneic patient in the office. *Prim Care.* 2006;33(3):643-657.
- 11. Salvi SS, Barnes PJ. Chronic obstructive pulmonary disease in non-smokers. *Lancet.* 2009;374(9691):733-743.
- Raghu G, Weycker D, Edelsberg J, Bradford WZ, Oster G. Incidence and prevalence of idiopathic pulmonary fibrosis. Am J Respir Crit Care Med. 2006;174(7):810-816.
- Pratter MR, Abouzgheib W, Akers S, Kass J, Bartter T. An algorithmic approach to chronic dyspnea. *Respir Med.* 2011;105(7):1014-1021.
- 14. National Heart, Lung, and Blood Institute. National Asthma Education and Prevention Program (NAEPP) Expert Panel Report 3: Guidelines for the Diagnosis and Management of Asthma Full Report. 2007. http://www.nhlbi. nih.gov/guidelines/asthma.
- Global Initiative for Chronic Obstructive Lung Disease. Global strategy for the diagnosis, management, and prevention of chronic obstructive lung disease. 2014. http://www.goldcopd.org/guidelines-global-strategy-for-diagnosismanagement.html.

- 16. Peters SP. When the chief complaint is (or should be) dyspnea in adults. *J Allergy Clin Immunol Pract.* 2013;1(2):129-136.
- Peters SP. Special considerations in adults for diagnoses that may coexist with or masquerade as asthma. Ann Allergy Asthma Immunol. 2010;104(6):455-560.
- American College of Cardiology Foundation, American Heart Association, European Society of Cardiology, et al. Management of patients with atrial fibrillation (compilation of 2006 ACCF/AHA/ESC and 2011 ACCF/AHA/ HRS recommendations): a report of the American College of Cardiology/ American Heart Association Task Force on practice guidelines. *Circulation*. 2013;127(18):1916-1926.
- Lubitz SA, Benjamin EJ, Ruskin JN, Fuster V, Ellinor PT. Challenges in the classification of atrial fibrillation. *Nat Rev Cardiol.* 2010;7(8):451-460.
- Katz PO, Gerson LB, Vela MF. Guidelines for the diagnosis and management of gastroesophageal reflux disease. Am J Gastroenterol. 2013;108(3):308-328.
- Gilbody S, Richards D, Brealey S, Hewitt C. Screening for depression in medical settings with the Patient Health Questionnaire (PHQ): a diagnostic metaanalysis. J Gen Intern Med. 2007;22(11):1596-1602.
- 22.Spitzer RL, Kroenke K, Williams JB, Löwe B. A brief measure for assessing generalized anxiety disorder: the GAD-7. Arch Intern Med. 2006;166(1):1092-1097.
- 23. Gidwani N, Louie S. Diagnosing and managing dyspnea. Consultant. 2014;54(2):129-130.

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