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14 Management of Stable Chronic Obstructive Pulmonary Disease

Key Points:
1. Before initiating management, establish the diagnosis of chronic obstructive pulmonary disease (COPD):
   - respiratory symptoms: cough, phlegm production, and/or breathlessness
   - documented airflow limitation by spirometry or
   - radiographic evidence of emphysema
2. The most recent GOLD guidelines base management upon 3 factors:
   - severity of airflow limitation measured by FEV₁
   - respiratory healthcare visits in the prior 12 months
   - symptom score measured by the COPD Assessment Test or breathlessness measured by the modified Medical Research Council scale
3. In general, COPD pharmacologic management begins with a short acting bronchodilator and progresses with the addition of a long acting bronchodilator (either an anticholinergic or a long acting beta agonist), and lastly an inhaled corticosteroid.
4. Nonpharmacologic interventions such as oxygen, vaccinations, and pulmonary rehabilitation are essential components to comprehensive COPD management.

14.1 Management of Stable COPD

The prerequisite for the optimal management of COPD is the ascertainment of the correct diagnosis; COPD is diagnosed by respiratory symptoms (usually cough, sputum production, and/or dyspnea), the presence of airflow limitation (FEV₁/FVC < lower limit of normal), and the exclusion of other processes (see Chapter 3, COPD Recognition and Diagnosis: Approach to the Patient with Respiratory Symptoms).

The goals of COPD management are multiple and diverse: 1) reduce mortality, 2) preserve lung function, 3) decrease COPD-associated complications, 4) treat COPD-related comorbidities, 5) decrease the number and severity of COPD exacerbations, 6) relieve respiratory symptoms, especially breathlessness and cough, and 7) improve overall wellbeing. Although exacerbation prevention, reduction in healthcare utilization, and preservation of lung function are frequently the endpoints of investigations examining the effects of pharmacologic and nonpharmacologic interventions in COPD, relief of respiratory symptoms is often the principle goal when a primary care provider sees a patient with COPD.

The major symptoms experienced by patients with COPD are breathlessness, cough, and sputum production (Rennard, 2002; Leidy, 2003A). Assessment of these
symptoms should be performed at every healthcare encounter for individuals with COPD. Numerous surveys and assessment tools have been developed to assist clinicians with the evaluation of respiratory symptoms. One such tool is the Breathlessness, Cough, and Sputum Scale, a daily diary of COPD related symptoms that correlates with lung function measured by the FEV₁ and sputum volume (Leidy, 2003). This scale is simple and consists of only three questions: 1) How much difficulty did you have breathing today?, 2) How was your cough today?, and 3) How much trouble was your sputum today? Each question has 5 graded responses from none to severe (Leidy, 2003A; Leidy, 2003B). This questionnaire is limited to the three major respiratory symptoms associated with COPD. Numerous other disease-specific, as well as generic surveys, have been developed to assess quality of life in individuals with COPD. In a systemic review, the best instruments were the Chronic Respiratory Questionnaire (CRQ), COPD Assessment Test (CAT), Saint George Respiratory Questionnaire (SGRQ), and Living with COPD questionnaire (LCOPD) (Weldam, 2012). The CAT has been adopted in the most recent GOLD guidelines (Jones, 2009) (Figure 14.1).

14.1.1 Breathlessness

Breathlessness at rest and especially with exertion is a primary symptom of COPD. Dyspnea often limits action or patients avoid activities due to the fear of developing breathlessness. The cause of breathlessness in individuals with COPD is multifactorial with contributions from bronchospasm, desaturation, secretions, and cough but the major factor is dynamic lung hyperinflation. Lung hyperinflation is an increase in the volume of air remaining in the lungs at the end of exhalation that is associated with excessive loading and functional weakness of the respiratory muscles that precipitates mechanical dysfunction and sensory dysphoria (the sensation of breathlessness). Lung hyperinflation is caused by increased lung compliance and airflow limitation. In COPD, air is easily inhaled but exhalation is impeded by airflow limitation caused by increased resistance and reduced elastic recoil; if less air is exhaled than was inhaled, the lung begins to retain air, increasing the end expiratory lung volume (EELV). As EELV increases, the volume of air inhaled during subsequent breaths is decreased due to restriction of the inspiratory capacity, reducing the tidal volume, and impairing minute ventilation. Thus, the lungs are unable to meet ventilatory and oxygenation demands. The increase in respiratory rate that occurs with exertion further augments hyperinflation by reducing expiratory time causing more air trapping and elevation of EELV. As hyperinflation increases, the respiratory muscles are stretched or loaded causing discomfort; the stretching also causes functional weakness by putting the muscles at a mechanical disadvantage. The discomfort caused by stretching and loading of respiratory muscles by dynamic hyperinflation is a significant factor contributing to the sensation of breathlessness. Both pharmacologic and non-pharmacologic treatments may help to ameliorate dynamic hyperinflation.
Pursed lip breathing facilitates exhalation of air by creating an increased expiratory resistance that elevates the intra-airway pressure to maintain airway patency, reducing collapse due to diminished elastic recoil and increasing the amount of air expelled during exhalation. The improved expiratory airflow reduces air trapping and hyperinflation. Slow, deliberate, and controlled breathing utilizing pursed lip breathing helps to reduce the respiratory rate which increases the exhalation time that may also reduce air trapping and hyperinflation. Control of anxiety, relaxation techniques, and better awareness of the perception of breathlessness may also help reduce the rate of breathing. Oxygen and improved overall fitness from pulmonary rehabilitation or other conditioning programs may reduce ventilatory demand slowing the respira-
Chronic bronchitis is defined as cough and sputum production for at least 3 months of the year for two consecutive years. Individuals with mild to moderate airflow limitation report cough and sputum production more frequently than those with severe obstruction (von Hertzen, 2000). Causes of cough in COPD have not been well studied but inflammation, sputum clearance, and comorbidities such as bronchiectasis, gastroesophageal reflux disease, and postnasal drainage may all contribute. Early studies suggested that patients with COPD had a heightened cough response to capsaicin that was similar to individuals with asthma but further studies have been equivocal (Doherty, 2000).

Patients with COPD and chronic cough have longer durations of smoking, more impaired lung function, and are more likely to be current smokers (Kanner, 1999). The prevalence of chronic cough decreases by 80% 5 years after quitting smoking. Productive cough is a risk factor for the development of airflow limitation in nonsmokers and former smokers (Yamane, 2010).
Patients with moderately severe COPD and cough spend more time coughing during the day than at night (Smith, 2003). Cough frequency does not correlate with the severity of airflow limitation (Smith, 2003; Smith, 2004). Despite the frequent reporting of cough by patients with COPD, objective cough counts and time spent coughing, 12.3 s per hour during the day and 1.63 s per hour at night, are quite low (Smith, 2003; Smith, 2004).

Inhaled steroids do not affect cough frequency in patients with COPD (Doherty, 2000; Smith, 2004). Long acting beta agonists reduce cough more effectively than short acting beta agonists (Smith, 2004); in contrast, short acting anticholinergics reduce cough better than long acting anticholinergics (Smith, 2004). Codeine is not better than placebo in reducing cough frequency in patients with COPD (Smith, 2006).

14.1.3 Sputum Production

Airway mucus is usually a thin gel composed of water and mucins (glycosylated proteins) as well as components of the innate immune system: antimicrobial, immunomodulatory, and protective proteins, that line the respiratory mucosal surface. Inhaled toxins and particles are trapped within the mucus and expelled from the lungs by ciliary beating and cough (Fahey, 2010). Cigarette smoke impairs cilia structure and function, induces mucin production, and reduces mucus hydration. Thus, more airway mucus remains in the lower airway and may become colonized with bacteria which can induce inflammation and inflammatory cell migration into the airway. Neutrophil-derived DNA increases the viscosity of the airway mucus further impairing clearance.

Patients with severe COPD frequently experience increased phlegm production and have more respiratory symptoms, less emphysema, and greater airflow limitation (Kim, 2011). Approximately 14% of individuals with airflow limitation have symptoms of chronic bronchitis and, compared with those with airflow limitation but no symptoms of chronic bronchitis, have worse lung function and overall health, more respiratory symptoms and exacerbations, greater limitations in physical activity, and an increased risk of mortality (Montes de Oca, 2012; Burgel, 2009; Kim, 2011). Most recently the COPDGene Study has shown that individuals with the chronic bronchitic phenotype of COPD have an accelerated rate of decline in lung function, increased risk of respiratory infections, more frequent nasal and respiratory symptoms, and increased frequency and severity of exacerbations (Kim, 2011).

Bronchiectasis is another contributing factor to cough and sputum production. Approximately 30–60% of individuals with COPD have chest CT evidence of bronchiectasis (Bafadhel, 2011; Martínez-García, 2011). The presence of bronchiectasis is associated with increased frequency of bacterial colonization of the lower airway, more severe airflow limitation, and previous hospitalizations for COPD exacerbations.
Smoking cessation is associated with a reduction in phlegm production (Kanner, 1999). Airway clearance techniques such as postural drainage, percussion, vibration, and breathing exercises do not reduce COPD exacerbations, have variable effects on lung function, improve quality of life, and may promote short term increases in phlegm production (Osadnik, 2012). Mucolytics (such as N-acetyl cysteine) reduce exacerbation rates in patients with chronic bronchitis by approximately 20% (Poole, 2012).

### 14.2 Guidelines for the Management of COPD

There are multiple guidelines for the management of COPD that have been developed by numerous national and international health organizations. The Global Initiative for Chronic Obstructive Lung Disease (GOLD) reports are the most widely disseminated international guidelines for the management of COPD and are continually updated (GOLD guidelines). Another source for practical advice for the management of COPD is the COPD Foundation Guide to COPD Diagnosis and Treatment (http://journal.copdfoundation.org/Portals/0/JCOPDF/Files/Issue1-Volume1/JCOPDF-2014-0124-Pocket-Guide.pdf).

COPD is a systemic inflammatory state with protean manifestations—COPD is not just a lung disease! (see Chapter 10. COPD Is a Multi-organ Disorder: Systemic Manifestations). Cardiovascular disease including coronary artery disease, stroke, and hypertension, osteoporosis, psychological disorders, especially anxiety and depression, skeletal muscle dysfunction, and lung cancer occur more frequently in individuals with COPD than in those with normal lung function even though they may have similar smoking histories. Treatment of COPD comorbidities should include optimal COPD management as well as appropriate management of the specific nonpulmonary process.

Most COPD treatment guidelines advocate an additive, incremental approach to the pharmacologic management of COPD. There has been poor adherence to most COPD guidelines and few studies have evaluated their effectiveness. At the Cincinnati VAMC, review of the prescribed respiratory medications and respiratory and nonrespiratory visits between 2000 and 2005 showed poor compliance with the GOLD pharmacologic treatment guidelines but correlation between respiratory treatments and the number of prior respiratory health care visits (Seaman, 2010). Another study from Switzerland showed that adherence to the GOLD guidelines did not affect respiratory symptoms, exacerbation rate, or lung function over 12 months (Jochmann, 2012). However, other studies have shown that better adherence with respiratory medications improves overall survival in individuals with COPD (Vestbo, 2009). Although these earlier guidelines classified disease impairment and based treatment recommendations upon the severity of airflow limitation (usually measured by the FEV1), the most recent 2012 GOLD guidelines include an assessment of respiratory symptoms and prior healthcare encounters to assist in the determination of the optimal treatment regimen.
14.2.1  GOLD Guidelines for the Management of COPD

The GOLD guidelines recommend that each individual with COPD be evaluated to determine the severity of COPD, the effect of COPD on the individual’s overall health and well being, the risk of future COPD-related events (healthcare encounters or death), and COPD-related comorbidities (Figure 14.3). COPD severity is classified by the severity of airflow limitation measured by spirometry. The GOLD guidelines define airflow limitation as FEV₁/FVC < 0.70 (see Chapter 4, Pulmonary Function Testing: Presence and Severity of Airflow Limitation/Obstruction).

The effects of COPD on an individual’s overall health and well being are evaluated by the COPD Assessment Test (CAT) (Figure 14.1) or the Modified British Medical Research Council (mMRC) breathlessness scale (Table 14.2).
Although there are numerous definitions of a COPD exacerbation (see Chapter 16, Management of Outpatient COPD Exacerbations and Chapter 17, Management of Inpatient COPD Exacerbations), the GOLD guidelines define a COPD exacerbation as an acute event characterized by worsening of the patient’s respiratory symptoms that is beyond normal day-to-day variations and leads to a change in medication (GOLD Guidelines). The best predictor of future exacerbations is a history of exacerbations in the prior year (Hurst, 2010). Severity of airflow limitation and use of systemic steroids are also associated with more frequent COPD exacerbations.

The GOLD multivariable COPD assessment is complex and requires several disparate pieces of data. It classifies patients as high or low risk based upon the severity of airflow limitation and history of exacerbations and then grades them as more or less symptomatic based upon their CAT or mMRC score (Figure 14.3). Patients with less symptoms, better spirometry, and fewer exacerbations are group A; patients with more symptoms, worse spirometry, and more frequent exacerbation are group D. Groups B and C are intermediate and have either more symptoms and lower risk or less symptoms and higher risk.

### Table 14.1: GOLD Classification of COPD Severity (GOLD Guidelines)

<table>
<thead>
<tr>
<th>GOLD Classification of Airflow Limitation Severity</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>GOLD 1 Mild</td>
<td>FEV&lt;sub&gt;1&lt;/sub&gt; ≥ 80% of predicted</td>
</tr>
<tr>
<td>GOLD 2 Moderate</td>
<td>50% &lt; FEV&lt;sub&gt;1&lt;/sub&gt; &lt; 80% of predicted</td>
</tr>
<tr>
<td>GOLD 3 Severe</td>
<td>30% &lt; FEV&lt;sub&gt;1&lt;/sub&gt; &lt; 50% of predicted</td>
</tr>
<tr>
<td>GOLD 4 Very Severe</td>
<td>FEV&lt;sub&gt;1&lt;/sub&gt; &lt; 30% of predicted</td>
</tr>
</tbody>
</table>

### Table 14.2: MMRC Dyspnea Scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description of Breathlessness</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>I only get breathless with strenuous exercise.</td>
</tr>
<tr>
<td>1</td>
<td>I get short of breath when hurrying on level ground or walking up a slight hill.</td>
</tr>
<tr>
<td>2</td>
<td>On level ground, I walk slower than people of the same age because of breathlessness, or have to stop for breath when walking at my own pace.</td>
</tr>
<tr>
<td>3</td>
<td>I stop for breath after walking about 100 yards or after a few minutes on level ground.</td>
</tr>
<tr>
<td>4</td>
<td>I am too breathless to leave the house or I am breathless when dressing.</td>
</tr>
</tbody>
</table>
Medications for the treatment of COPD consist of short acting beta agonists (SABA), long acting beta agonists (LABA), short acting anticholinergics (SAch), long acting anticholinergics (LAch), inhaled (ICS) and enteral corticosteroids. Other medications include phosphodiesterase inhibitors, macrolide antibiotics (used for their anti-inflammatory not antibiotic effect), and mucolytics.

SABAs are usually the initial medication and should be used on an as needed rather than scheduled basis for patients in GOLD group A. (Figure 14.4) Combinations of SABA and SAch (such as Combivent®) should not be prescribed regularly because SABA’s should only be used on an as needed basis. If symptoms persist despite use of a SABA or if SABA use is excessive (greater than 3–4 times daily), an anticholinergic bronchodilator is usually added, GOLD Group B. LAch’s are usually preferred because of the ease of use and theoretical advantage of improved adherence with less fre-
quent dosing. A LABA may also be added. Combinations of a LABA and ICS or LABA and LACH may facilitate medication use and improve adherence, GOLD group C. ICS are recommended for patients with GOLD severe or very severe disease (FEV1<50%) and who have had two or more exacerbations in the prior year, GOLD Group D. ICS may also be beneficial in individuals who demonstrate bronchodilator responsiveness during spirometry testing (Chapter 9.4, COPD Phenotypes). Use of triple inhalers in COPD, LACH and LABA plus ICS, improves lung function and symptoms but does not reduce exacerbations compared with either treatment alone (Aaron, 2007).

Many patients with less severe and/or less COPD-related risk are prescribed ICS. It has been estimated that 70% of patients with COPD are treated with high-dose combination inhalers yet only 10% qualify under the current guidelines (Barnes, 2011). Similarly, significant over prescription of ICS compared with guidelines was noted among Veterans with COPD at the Cincinnati VAMC (Seaman, 2010). Use of ICS is associated with an increased risk of pneumonia in patients with COPD, OR 1.78 (95% CI, 1.50–2.12) (Kew, 2014).

Over the past 5 years, numerous new medications and novel medication combinations for the management of COPD have been approved by the US Food and Drug Administration (Table 14.3). The pharmacologic armamentarium for the treatment of COPD has doubled in the past several years and many new medications and combinations are in development.

14.3.1 Phosphodiesterase Inhibitors

Phosphodiesterases (PDEs) are a family of at least 11 isoenzymes that hydrolyze cAMP and cGMP. Inhibition of PDEs stimulates bronchodilation and also reduces pulmonary inflammation. Methylxanthines such as aminophylline or theophylline are non-specific PDE inhibitors that have been used for the treatment of asthma and COPD. These medications are limited by a narrow therapeutic serum drug range, frequent interaction with other medications, and poor tolerability. Most recently, specific inhibitors of PDE isoforms, especially PDE4, have been developed and shown to be effective in the management of COPD. Roflumilast was approved by the FDA in 2011 as adjuvant treatment to reduce COPD exacerbations in patients with severe COPD associated with chronic bronchitis and a history of exacerbations (Reid, 2012).

14.3.2 Macrolides

Macrolides such as erythromycin and azithromycin have anti-inflammatory properties in addition to anti-microbial effects. Recent studies have shown that either daily erythromycin or azithromycin decrease the frequency of COPD exacerbations in patients with a history of exacerbations. (Martinez, 2008; Seemungal, 2008; Albert,
Table 14.3: Federal Drug Administration Approved Medications for COPD

<table>
<thead>
<tr>
<th>Class</th>
<th>Generic Name</th>
<th>Brand Name</th>
<th>FDA Approval</th>
</tr>
</thead>
<tbody>
<tr>
<td>SABA</td>
<td>Albuterol</td>
<td>Ventolin HFA</td>
<td>2001</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Proventil HFA</td>
<td>1999</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Xopenex</td>
<td>2002</td>
</tr>
<tr>
<td>SACH</td>
<td>Ipratropium</td>
<td>Atrovent HFA</td>
<td>2004</td>
</tr>
<tr>
<td>SABA + SACH</td>
<td>albuterol + ipratropium</td>
<td>Duoneb</td>
<td>2001</td>
</tr>
<tr>
<td>LACH</td>
<td>Tiotropium bromide</td>
<td>Spiriva HandiHaler</td>
<td>2004</td>
</tr>
<tr>
<td></td>
<td>Umeclidinium</td>
<td>Incruse Ellipta</td>
<td>2014</td>
</tr>
<tr>
<td></td>
<td>Aclidinium bromide</td>
<td>Tudorza Pressair</td>
<td>2012</td>
</tr>
<tr>
<td>LABA</td>
<td>Formoterol fumarate</td>
<td>Foradil Aerolizer</td>
<td>2001</td>
</tr>
<tr>
<td></td>
<td>Salmeterol</td>
<td>Serevent Diskus</td>
<td>1998</td>
</tr>
<tr>
<td></td>
<td>Indacaterol maleate</td>
<td>Arcapta</td>
<td>2011</td>
</tr>
<tr>
<td></td>
<td>Arformoterol tartrate</td>
<td>Brovana</td>
<td>2006</td>
</tr>
<tr>
<td></td>
<td>Olodaterol</td>
<td>Stiverdi Respimat</td>
<td>2014</td>
</tr>
<tr>
<td>LABA + ICS</td>
<td>Formoterol + Budesonide</td>
<td>Symbicort</td>
<td>2009</td>
</tr>
<tr>
<td></td>
<td>Salmeterol + Fluticasone furoate</td>
<td>Advair Diskus</td>
<td>2003</td>
</tr>
<tr>
<td></td>
<td>Vilanterol + Fluticasone furoate</td>
<td>Breo Ellipta</td>
<td>2013</td>
</tr>
<tr>
<td>LACH + LABA</td>
<td>Umeclidinium + vilanterol</td>
<td>Anoro Ellipta</td>
<td>2013</td>
</tr>
<tr>
<td>PDE 4 Inhibitor</td>
<td>Roflumilast</td>
<td>Daliresp</td>
<td>2011</td>
</tr>
</tbody>
</table>

Abbreviations: SABA: short acting bronchodilator; SACH: short acting anticholinergic; LACH: long acting anticholinergic; LABA: long acting bronchodilator; ICS: inhaled corticosteroid; PDE: phosphodiesterase

2011) Although it remains unclear which subgroup of patients with COPD will benefit best from macrolide treatment and whether dosing should be daily or thrice weekly, current recommendations are to consider daily macrolide treatment in patients who, despite maximal standard bronchodilator therapy, have at least 2 exacerbations yearly (Mammen, 2012). Potential adverse effects of prolonged macrolide use include development of bacterial resistance, cardiovascular events, and hearing loss.
14.3.3 Mucolytics

Mucolytics such as n-acetylcysteine and carbocysteine may reduce COPD exacerbations and improve health related quality of life in patients with COPD (Decramer, 2010). In one prospective study, n-acetylcysteine reduced hyperinflation (Stav, 2009).

14.3.4 Supplemental Oxygen

Supplemental oxygen improves survival in patients with hypoxemia at rest (PaO₂ <55 torr or SpO₂ <88%; or PaO₂ < 60 and >55 torr with evidence of cor pulmonale). The mechanism(s) by which supplemental oxygen improves mortality is not known. Oxygenation should be measured on room air at rest, with exertion, and during sleep after the administration of supplemental oxygen to insure that desaturation is prevented. Although Medicare and most insurances reimburse for supplemental oxygen during exercise or at night with evidence of exercise or nocturnal desaturation, there is no substantive evidence that supplemental oxygen during exercise or at night is beneficial in individuals with stable COPD and normoxemia at rest.

14.4 Nonpharmacologic Treatment of COPD

*Smoking cessation* is the singularly most important intervention for the prevention and treatment of COPD. Please see Chapter 7, Smoking Cessation, for further discussion of smoking cessation.

*Both influenza and pneumococcal vaccination* are recommended for individuals with COPD. Influenza vaccination reduces mortality, outpatient visits, hospitalizations, and exacerbations caused by influenza (Varkey, 2009). In contrast, although pneumococcal vaccination reduces the incidence of invasive pneumococcal disease, it has not shown any significant effect on mortality, rates of pneumonia or exacerbations, lung function, or cost effectiveness (Varkey, 2009; Walters, 2010). Vaccination against both influenza and pneumococcus may reduce COPD exacerbations more effectively than either vaccine alone (Varkey, 2009).

*Pulmonary rehabilitation* is a multidisciplinary program of education and exercise that teaches patients with COPD about their disease, its treatment, and mechanisms to cope with its consequences as well as an exercise and conditioning program. Pulmonary rehabilitation has the best effect when it is integrated into a comprehensive COPD management program that encourages behavior change and a shift from provider initiated to patient initiated care. Patients with COPD who maintain physical activity have less breathlessness with exertion, better health related quality of life, improved long term function and independence, and better psychological and physiological function.
Physical inactivity is associated with worse survival, increased risk of respiratory-related hospitalization, lower self-reported health status, and greater systemic inflammation (Watz, 2014). Pulmonary rehabilitation improves respiratory symptoms (Lacasse, 2006). In addition to improving respiratory symptoms, pulmonary rehabilitation decreases health care utilization and may improve survival (Ries, 2008). Home based PR programs have the equivalent benefit of hospital based programs (Bourbeau, 2010).

**Lung volume reduction** either surgical or endoscopic via endobronchial valve placement may also be effective in certain patients with COPD. Surgical lung volume reduction improves exercise tolerance, quality of life, and survival in selected patients with COPD (Tidwell, 2012). LVRS is only beneficial in patients with upper lobe emphysema and poor exercise tolerance and is detrimental in individuals with FEV$_1$ < 20%.

### Table 14.4: Medicare Oxygen Reimbursement Guidelines

<table>
<thead>
<tr>
<th>Patient Condition</th>
<th>PaO$_2$</th>
<th>SpO$_2$</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Awake, at rest</strong></td>
<td>less than 55 mm Hg or 56–59 mm Hg and dependent edema or cor pulmonale or pulmonary hypertension by right heart catheterization or ECHO or P pulmonale on ECG (P wave &gt; 3 mm in leads II, III, or AVF) or erythrocythemia (HCT&gt; 56%)</td>
<td>less than 88% or = 89% and dependent edema or cor pulmonale or pulmonary hypertension by right heart catheterization or ECHO or P pulmonale on ECG (P wave &gt; 3 mm in leads II, III, or AVF) or erythrocythemia (HCT&gt; 56%)</td>
</tr>
<tr>
<td><strong>Exercise</strong></td>
<td>less than 55 mm Hg and Documentation that use of supplemental oxygen ameliorates the decline in oxygen levels (Duration of desaturation and type/level of exertion are not specified.)</td>
<td>less than 88% mm Hg and Documentation that use of supplemental oxygen ameliorates the decline in oxygen levels (Duration of desaturation and type/level of exertion are not specified.)</td>
</tr>
<tr>
<td><strong>Sleep</strong></td>
<td>less than 55 mm Hg or PaO$_2$ declines &gt; 10 mm Hg from awake, resting level and dependent edema or cor pulmonale or pulmonary hypertension by right heart catheterization or ECHO or P pulmonale on ECG (P wave &gt; 3 mm in leads II, III, or AVF) or erythrocythemia (HCT&gt; 56%) (Duration of desaturation and type/level of exertion are not specified.)</td>
<td>less than 88% or SpO$_2$ declines &gt; 5% from awake, resting level and dependent edema or cor pulmonale or pulmonary hypertension by right heart catheterization or ECHO or P pulmonale on ECG (P wave &gt; 3 mm in leads II, III, or AVF) or erythrocythemia (HCT&gt; 56%) (Duration of desaturation and type/level of exertion are not specified.)</td>
</tr>
</tbody>
</table>
of predicted, DLCO < 20% of predicted, or diffusely distributed emphysema. Patients who are being considered for LVRS should be referred for pulmonary consultation. Endoscopic LVRS is a newer technique that may also be beneficial in select patients.

### 14.5 Management of COPD-related Nonpulmonary Co-morbidities

Because COPD is a multisystemic disorder with protean nonpulmonary manifestations, it is essential to evaluate and treat patients with COPD for conditions such as anemia, diabetes, lung cancer, and cardiovascular disease. (Reviewed in Chapter 10, COPD Is a Multi-Organ Disorder: Systemic Manifestations.)

### 14.6 Summary Points

1. Establish a definitive diagnosis of COPD before initiating treatment.
2. The principal symptoms of COPD are breathlessness, cough, and sputum production.
3. There are several well validated instruments (such as the COPD Activity Test) to assess and monitor serially COPD’s effects on quality of life and respiratory symptoms.
4. Pharmacologic treatment for COPD begins with an as needed short acting bronchodilator, usually a SABA. The subsequent medication is a long acting bronchodilator, either LACH or LABA. Next, the other class of long acting bronchodilator is added and inhaled corticosteroids are usually the final inhaler category. (Individuals with COPD who have a response to bronchodilators may be more responsive to inhaled corticosteroids.)
5. Recent definitions of new COPD phenotypes will help to individualize COPD treatment regimens.
6. Dynamic hyperinflation is the major cause of exertional dyspnea and responds to pursed lip breathing and a slowing of the respiratory rate.
7. Nonpharmacologic treatments that are effective in the management of COPD include supplemental oxygen, lung volume reduction surgery, pulmonary rehabilitation and vaccinations, especially influenza.

### References

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References


