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# At-A-Glance Outpatient Management Reference for Chronic Obstructive Pulmonary Disease (COPD)



**BASED ON THE GLOBAL STRATEGY FOR DIAGNOSIS,  
MANAGEMENT AND PREVENTION OF COPD  
GLOBAL INITIATIVE FOR CHRONIC OBSTRUCTIVE LUNG DISEASE (GOLD)  
2017 REPORT**

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Please refer to the 2017 GOLD Report at [www.goldcopd.org](http://www.goldcopd.org)

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## DIAGNOSING COPD

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COPD should be considered in any patient who has dyspnea, chronic cough or sputum production, and/or a history of exposure to risk factors for the disease (Table 2.1).\*

Table 2.1. Key indicators for considering a diagnosis of COPD	
<i>Consider COPD, and perform spirometry, if any of these indicators are present in an individual over age 40. These indicators are not diagnostic themselves, but the presence of multiple key indicators increases the probability of a diagnosis of COPD. Spirometry is required to establish a diagnosis of COPD.</i>	
Dyspnea that is:	Progressive over time. Characteristically worse with exercise. Persistent.
Chronic cough:	May be intermittent and may be unproductive. Recurrent wheeze.
Chronic sputum production:	Any pattern of chronic sputum production may indicate COPD.
Recurrent lower respiratory tract infections	
History of risk factors:	
	Host factors (such as genetic factors, congenital/developmental abnormalities etc). Tobacco smoke (including popular local preparations). Smoke from home cooking and heating fuels. Occupational dusts, vapors, fumes, gases and other chemicals.
Family history of COPD and/or childhood factors:	
	For example low birthweight, childhood respiratory infections etc.

**Spirometry is required to make the diagnosis of COPD in this clinical context;** the presence of a post-bronchodilator  $FEV_1/FVC < 0.70$  confirms the presence of persistent airflow limitation and thus of COPD in patients with appropriate symptoms and significant exposures to noxious stimuli.

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## ASSESSMENT OF COPD

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The goals of COPD assessment are to determine the level of airflow limitation, its impact on the patient's health status and the risk of future events (such as exacerbations, hospital admissions or death), in order to, eventually, guide therapy.

To achieve these goals, COPD assessment must consider the following aspects

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\* Please note that table & figure numbers are retained from the full GOLD 2017 report to facilitate cross referencing.

of the disease separately:

- The presence and severity of the spirometric abnormality
- Current nature and magnitude of the patient's symptoms
- Exacerbation history and future risk
- Presence of comorbidities

Table 2.4. Classification of airflow limitation severity in COPD (Based on post-bronchodilator FEV <sub>1</sub> )		
In patients with FEV <sub>1</sub> /FVC < 0.70:		
GOLD 1:	Mild	FEV <sub>1</sub> ≥ 80% predicted
GOLD 2:	Moderate	50% ≤ FEV <sub>1</sub> < 80% predicted
GOLD 3:	Severe	30% ≤ FEV <sub>1</sub> < 50% predicted
GOLD 4:	Very Severe	FEV <sub>1</sub> < 30% predicted

**Assess degree of airflow limitation using spirometry:** The classification of airflow limitation severity in COPD is shown in **Table 2.4**. Specific spirometric cut-points are used for purposes of simplicity. Spirometry should be performed after the administration of an adequate dose of at least one short-acting inhaled bronchodilator in order to minimize variability.

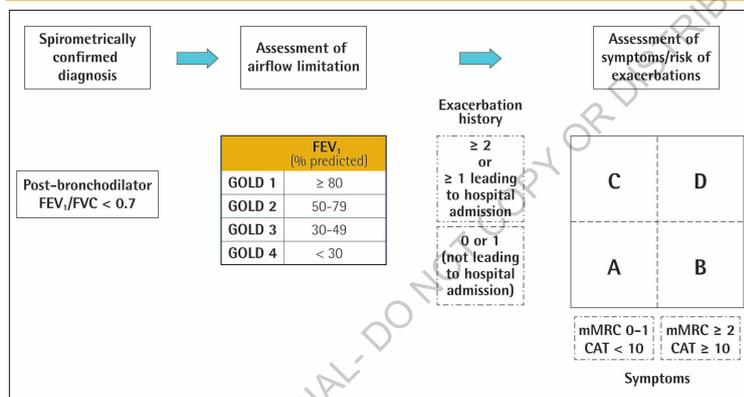
**Assess symptoms:** a comprehensive assessment of symptoms is recommended rather than just a measure of breathlessness. The COPD Assessment Test (CAT™) and The COPD Control Questionnaire (The CCQ®) have been developed and are suitable.

**Assess exacerbation risk:** COPD exacerbations are defined as an acute worsening of respiratory symptoms that result in additional therapy. These events are classified as mild (treated with short acting bronchodilators (SABDs) only), moderate (treated with SABDs plus antibiotics and/or oral corticosteroids) or severe (patient requires hospitalization or visits the emergency room). Severe exacerbations may also be associated with acute respiratory failure.

**Assess comorbidities:** Common comorbidities include cardiovascular disease, skeletal muscle dysfunction, metabolic syndrome, osteoporosis, depression, anxiety and lung cancer. The existence of COPD may increase the risk for other diseases such as lung cancer.

**Revised combined COPD assessment tool:** ABCD groups will now be derived exclusively from patient symptoms and their history of exacerbation. Spirometry, in conjunction with patient symptoms and exacerbation history, remains vital for the diagnosis, prognostication and consideration of other important therapeutic approaches. This new approach to assessment is illustrated in **Figure 2.4**.

Figure 2.4. The refined ABCD assessment tool



## PREVENTION OF COPD

**Smoking cessation** has the greatest capacity to influence the natural history of COPD. If effective resources and time are dedicated to smoking cessation, long-term quit success rates of up to 25% can be achieved (**Table 3.1**).

**Influenza vaccination** can reduce serious illness (such as lower respiratory tract infections requiring hospitalization) and death in COPD patients.

**Pneumococcal vaccinations**, PCV13 and PPSV23, are recommended for all patients ≥ 65 years of age.

**Identification and reduction of exposure to risk factors** is important in the treatment and prevention of COPD.

Table 3.1. Brief strategies to help the patient willing to quit	
• <b>ASK:</b>	Systematically identify all tobacco users at every visit. <i>Implement an office-wide system that ensures that, for EVERY patient at EVERY clinic visit, tobacco-use status is queried and documented.</i>
• <b>ADVISE:</b>	Strongly urge all tobacco users to quit. <i>In a clear, strong, and personalized manner, urge every tobacco user to quit.</i>
• <b>ASSESS:</b>	Determine willingness and rationale of patient's desire to make a quit attempt. <i>Ask every tobacco user if he or she is willing to make a quit attempt at this time (e.g., within the next 30 days).</i>
• <b>ASSIST:</b>	Aid the patient in quitting. <i>Help the patient with a quit plan; provide practical counseling; provide intra-treatment social support; help the patient obtain extra-treatment social support; recommend use of approved pharmacotherapy except in special circumstances; provide supplementary materials.</i>
• <b>ARRANGE:</b>	Schedule follow-up contact. <i>Schedule follow-up contact, either in person or via telephone.</i>

## MANAGEMENT OF STABLE COPD

Once COPD has been diagnosed, effective management should be based on an individualized assessment to reduce both current symptoms and future risks of exacerbations (**Table 4.1**).

Table 4.1. Goals for treatment of stable COPD	
<ul style="list-style-type: none"> <li>• Relieve symptoms</li> <li>• Improve exercise tolerance</li> <li>• Improve health status</li> </ul>	<p>REDUCE SYMPTOMS</p>
and	
<ul style="list-style-type: none"> <li>• Prevent disease progression</li> <li>• Prevent and treat exacerbations</li> <li>• Reduce mortality</li> </ul>	<p>REDUCE RISK</p>

**Pharmacologic therapies** can reduce symptoms, and the risk and severity of exacerbations, as well as improve health status and exercise tolerance. The classes of medications commonly used in treating COPD are shown in **Table 3.3**.

Table 3.3. Commonly used maintenance medications in COPD*					
Drug	Inhaler (mcg)	Solution for nebulizer (mg/ml)	Oral	Vials for injection (mg)	Duration of action (hours)
<b>Beta<sub>2</sub>-agonists</b>					
<i>Short-acting</i>					
Fenoterol	100-200 (MDI)	1	2.5 mg (pill), 0.05% (syrup)		4-6
Levalbuterol	45-90 (MDI)	0.1, 0.21, 0.25, 0.42			6-8
Salbutamol (albuterol)	90, 100, 200 (MDI & DPI) <sup>†</sup>	1, 2, 2.5, 5 mg/ml	2, 4, 5 mg (pill), 8 mg (extended release tablet) 0.024%/0.4 mg (syrup)	0.1, 0.5 mg	4-6, 12 (ex- tended release)
Terbutaline	500 (DPI)		2.5, 5 mg (pill)	0.2, 0.25, 1 mg	4-6
<i>Long-acting</i>					
Arformoterol		0.0075 <sup>‡</sup>			12
Formoterol	4.5-9 (DPI)	0.01 <sup>†</sup>			12
Indacaterol	75-300 (DPI)				24
Olodaterol	2.5, 5 (SMI)				24
Salmeterol	25-50 (MDI & DPI)				12
<b>Anticholinergics</b>					
<i>Short-acting</i>					
Ipratropium bromide	20, 40 (MDI)	0.2			6-8
Oxipropium bromide	100 (MDI)				7-9
<i>Long-acting</i>					
Aclidinium bromide	400 (DPI), 400 (MDI)				12
Glycopyrronium bromide	15.6 & 50 (DPI) <sup>†</sup>		1 mg (solution)	0.2 mg	12-24
Tiotropium	18 (DPI), 2.5 & 5 (SMI)				24
Umeclidinium	62.5 (DPI)				24
<b>Combination of short-acting beta<sub>2</sub>-agonist plus anticholinergic in one device</b>					
Fenoterol/ipratropium	50/20 (SMI)	1.25, 0.5 mg in 4ml			6-8
Salbutamol/ipratropium	100/20 (SMI), 75/15 (MDI)	0.5, 2.5 mg in 3ml			6-8
<b>Combination of long-acting beta<sub>2</sub>-agonist plus anticholinergic in one device</b>					
Formoterol/aclidinium	12/400 (DPI)				12
Formoterol/glycopyrronium	9.6/18 (MDI)				12
Indacaterol/glycopyrronium	27.5/15.6 & 110/50 (DPI) <sup>†</sup>				12-24
Vilanterol/umeclidinium	25/62.5 (DPI)				24
Olodaterol/tiotropium	5/5 (SMI)				24
<b>Methylxanthines</b>					
Aminophylline			105 mg/ml (solution)	250, 500 mg	Variable, up to 24
Theophylline (SR)			100-600 mg (pill)	250, 400, 500 mg	Variable, up to 24
<b>Combination of long-acting beta<sub>2</sub>-agonist plus corticosteroids in one device</b>					
Formoterol/ beclomethasone	6/100 (MDI & DPI)				
Formoterol/budesonide	4.5/160 (MDI), 4.5/80 (MDI), 9/320 (DPI), 9/160 (DPI)				
Formoterol/mometasone	10/200, 10/400 (MDI)				
Salmeterol/fluticasone	5/100, 50/250, 5/500 (DPI), 21/45, 21/115, 21/230 (MDI)				
Vilanterol/fluticasone furoate	25/100 (DPI)				
<b>Phosphodiesterase-4 inhibitors</b>					
Roflumilast			500 mcg (pill)		

MDI – metered-dose inhaler; DPI – dry powder inhaler; SMI – soft mist inhaler  
<sup>\*</sup> Not all formulations are available in all countries; in some countries other formulations and dosages may be available  
<sup>†</sup> Dose availability varies by country  
<sup>‡</sup> Formoterol nebulized solution is based on the unit dose vial containing 20 mg in a volume of 2.0 ml  
<sup>§</sup> Dose varies by country

Proper inhaler technique is of high relevance (**Table 4.4** and **4.5**). Key points for the use of anti-inflammatory agents are summarized in **Table 4.6** and key points for the use of other pharmacologic treatments are summarized in **Table 4.7**.

<b>Table 4.4. Key points for inhalation of drugs</b>
<ul style="list-style-type: none"> <li>• The choice of inhaler device has to be individually tailored and will depend on access, cost, prescriber, and most importantly, patient's ability and preference.</li> <li>• It is essential to provide instructions and to demonstrate the proper inhalation technique when prescribing a device, to ensure that inhaler technique is adequate and re-check at each visit that patients continue to use their inhaler correctly.</li> <li>• Inhaler technique (and adherence to therapy) should be assessed before concluding that the current therapy requires modification.</li> </ul>

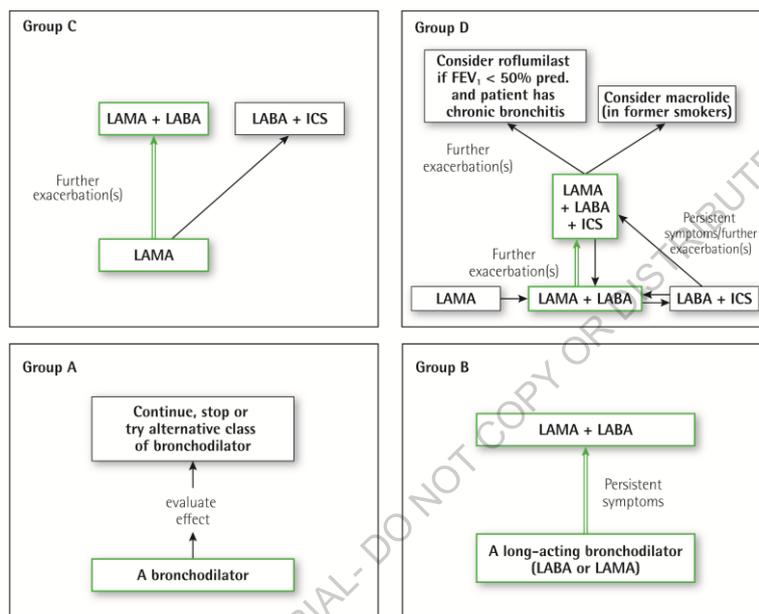
<b>Table 4.5. Key points for the use of bronchodilators</b>
<ul style="list-style-type: none"> <li>• LABAs and LAMAs are preferred over short-acting agents except for patients with only occasional dyspnea. (<b>Evidence A</b>).</li> <li>• Patients may be started on single long-acting bronchodilator therapy or dual long-acting bronchodilator therapy. In patients with persistent dyspnea on one bronchodilator treatment should be escalated to two. (<b>Evidence A</b>).</li> <li>• Inhaled bronchodilators are recommended over oral bronchodilators (<b>Evidence A</b>).</li> <li>• Theophylline is not recommended unless other long-term treatment bronchodilators are unavailable or unaffordable (<b>Evidence B</b>).</li> </ul>

<b>Table 4.6. Key points for the use of anti-inflammatory agents</b>
<ul style="list-style-type: none"> <li>• Long-term monotherapy with ICS is not recommended (<b>Evidence A</b>).</li> <li>• Long-term treatment with ICS may be considered in association with LABAs for patients with a history of exacerbations despite appropriate treatment with long-acting bronchodilators (<b>Evidence A</b>).</li> <li>• Long-term therapy with oral corticosteroids is not recommended (<b>Evidence A</b>).</li> <li>• In patients with exacerbations despite LABA/ICS or LABA/LAMA/ICS, chronic bronchitis and severe to very severe airflow obstruction, the addition of a PDE4 inhibitor can be considered (<b>Evidence B</b>).</li> <li>• In former smokers with exacerbations despite appropriate therapy, macrolides can be considered (<b>Evidence B</b>).</li> <li>• Statin therapy is not recommended for prevention of exacerbations (<b>Evidence A</b>).</li> <li>• Antioxidant mucolytics are recommended only in selected patients (<b>Evidence A</b>).</li> </ul>

<b>Table 4.7. Key points for the use of other pharmacological treatments</b>
<ul style="list-style-type: none"> <li>• Patients with severe hereditary alpha-1 antitrypsin deficiency and established emphysema may be candidates for alpha-1 antitrypsin augmentation therapy (<b>Evidence B</b>).</li> <li>• Antitussives cannot be recommended (<b>Evidence C</b>).</li> <li>• Drugs approved for primary pulmonary hypertension are not recommended for patients with a pulmonary hypertension secondary to COPD (<b>Evidence B</b>).</li> <li>• Low-dose long acting oral and parenteral opioids may be considered for treating dyspnea in COPD patients with severe disease (<b>Evidence B</b>).</li> </ul>

A proposed model for the initiation, and then subsequent escalation and/or de-escalation of pharmacologic management of COPD according to the individualized assessment of symptoms and exacerbation risk is shown in **Figure 4.1**.

Figure 4.1. Pharmacologic treatment algorithms by GOLD Grade [highlighted boxes and arrows indicate preferred treatment pathways]



Preferred treatment =   
 In patients with a major discrepancy between the perceived level of symptoms and severity of airflow limitation, further evaluation is warranted.

**Self-management education and coaching** by healthcare professionals should aim to motivate, engage and coach the patients to positively adapt their health behavior(s) and develop skills to better manage their disease.

Other non-pharmacological treatments are outlined in **Table 4.9**.

**Routine follow-up of COPD patients is essential.** Lung function may worsen over time, even with the best available care. Symptoms, exacerbations and objective measures of airflow limitation should be monitored to determine when to modify management and to identify any complications and/or comorbidities that may develop.

<b>Table 4.9. Key points for the use of non-pharmacological treatments</b>
<b>Education, self-management and pulmonary rehabilitation</b>
<ul style="list-style-type: none"> <li>• Education is needed to change patient's knowledge but there is no evidence that used alone it will change patient behavior.</li> <li>• Education self-management with the support of a case manager with or without the use of a written action plan is recommended for the prevention of exacerbation complications such as hospital admissions (<b>Evidence B</b>).</li> <li>• Rehabilitation is indicated in all patients with relevant symptoms and/or a high risk for exacerbation (<b>Evidence A</b>).</li> <li>• Physical activity is a strong predictor of mortality (<b>Evidence A</b>). Patients should be encouraged to increase the level of physical activity although we still don't know how to best insure the likelihood of success.</li> </ul>
<b>Vaccination</b>
<ul style="list-style-type: none"> <li>• Influenza vaccination is recommended for all patients with COPD (<b>Evidence A</b>).</li> <li>• Pneumococcal vaccination: the PCV13 and PPSV23 are recommended for all patients &gt; 65 years of age, and in younger patients with significant comorbid conditions including chronic heart or lung disease (<b>Evidence B</b>).</li> </ul>
<b>Nutrition</b>
<ul style="list-style-type: none"> <li>• Nutritional supplementation should be considered in malnourished patients with COPD (<b>Evidence B</b>).</li> </ul>
<b>End of life and palliative care</b>
<ul style="list-style-type: none"> <li>• All clinicians managing patients with COPD should be aware of the effectiveness of palliative approaches to symptom control and use these in their practice (<b>Evidence D</b>).</li> <li>• End of life care should include discussions with patients and their families about their views on resuscitation, advance directives and place of death preferences (<b>Evidence D</b>).</li> </ul>
<b>Treatment of hypoxemia</b>
<ul style="list-style-type: none"> <li>• In patients with severe resting hypoxemia long-term oxygen therapy is indicated (<b>Evidence A</b>).</li> <li>• In patients with stable COPD and resting or exercise-induced moderate desaturation, long term oxygen treatment should not be routinely prescribed. However, individual patient factors may be considered when evaluating the patient's needs for supplemental oxygen (<b>Evidence A</b>).</li> <li>• Resting oxygenation at sea level does not exclude the development of severe hypoxemia when travelling by air (<b>Evidence C</b>).</li> </ul>
<b>Treatment of hypercapnia</b>
<ul style="list-style-type: none"> <li>• In patients with severe chronic hypercapnia and a history of hospitalization for acute respiratory failure, long term non-invasive ventilation may be considered (<b>Evidence B</b>).</li> </ul>
<b>Intervention bronchoscopy and surgery</b>
<ul style="list-style-type: none"> <li>• Lung volume reduction surgery should be considered in selected patients with upper-lobe emphysema (<b>Evidence A</b>).</li> <li>• Bronchoscopic lung volume reduction interventions may be considered in selected patients with advanced emphysema (<b>Evidence B</b>).</li> <li>• In selected patients with a large bulla surgical bullectomy may be considered (<b>Evidence C</b>).</li> <li>• In patients with very severe COPD (progressive disease, BODE score of 7 to 10, and not candidate for lung volume reduction) lung transplantation may be considered for referral with at least one of the following: (1) history of hospitalization for exacerbation associated with acute hypercapnia (<math>P_{CO_2} &gt; 50</math> mm Hg); (2) pulmonary hypertension and/or cor pulmonale, despite oxygen therapy; or (3) <math>FEV_1 &lt; 20\%</math> and either <math>DLCO &lt; 20\%</math> or homogenous distribution of emphysema (<b>Evidence C</b>).</li> </ul>

