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Management of chronic obstructive pulmonary disease (COPD) – with a focus on the 2017 GOLD Strategy update

About the Expert

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Abbreviations used in this Review

ACOS = asthma-COPD overlap syndrome
COPD = chronic obstructive pulmonary disease
FEV_1 = forced expiratory volume in one second
FVC = forced vital capacity
ICS = inhaled corticosteroids
LABA = long-acting β-agonist
LAMA = long-acting muscarinic antagonist
MDI = metered-dose inhaler
RCT = randomised controlled trial
SABA = short-acting β-agonist
SABD = short-acting bronchodilator
SAMA = short-acting muscarinic antagonist
WHO = World Health Organisation

This publication is intended as an educational resource for primary healthcare professionals managing patients with chronic obstructive pulmonary disease (COPD). It follows on from our 2016 publication discussing the management of COPD in general practice and provides an update on the latest international recommendations for the treatment of this condition. The Global Initiative for Chronic Obstructive Lung Disease (GOLD) recently released their 2017 report on the diagnosis, management and prevention of COPD.[1] This is the 4th GOLD update based on a comprehensive review of new and relevant information published on COPD from 2015 to 2016. Spirometry remains necessary for accurate diagnosis and subsequent progression. The GOLD 2017 update gives greater focus to symptoms through the use of quality of life assessment tools and the effect of symptom burden on the life of COPD patients.

In brief, the changes to the 2017 GOLD strategy include:[1]

- Lung function (determined via spirometry) remains the gold standard for establishing diagnosis and prognosis but respiratory symptoms and exacerbation history alone are used to assign position on the GOLD ABCD treatment classification grid
- Greater emphasis is placed on individualised assessment and individualised management choices that incorporate self-management plans, pulmonary rehabilitation, integrative care including palliative care and 'end-of-life care'
- Greater guidance on personalised treatment options, with escalation and de-escalation strategies now suggested
- More detailed hospital discharge and follow-up criteria are outlined inclusive of management strategies of co-morbidities such as cardiovascular disease, including the vexing issues of multi-morbidity and polypharmacy
- Greater emphasis on the use of LAMA/LABA for appropriate patients
- Triple therapy is recommended for patients who have a high burden of symptoms and/or exacerbations despite initial maintenance therapy with dual inhalers
- ICS/LABAs are recommended for patients (1) with coexisting asthma or ACOS, (2) with a higher blood eosinophil count, and (3) where patients are unable to access newer treatment classes
- Recognition that there is a significant relationship between poor inhaler technique and symptom control in patients with COPD and that inhaler technique needs to be assessed regularly.

Introduction

COPD is a common preventable and treatable chronic respiratory disease characterised by persistent respiratory symptoms and airflow limitation that is not fully reversible.[1] This chronic airflow limitation is usually progressive and is caused by a mixture of parenchymal tissue destruction (emphysema) and small airways disease (obstructive bronchiolitis), usually occurring as an abnormal inflammatory response of the lungs to noxious particles or gases, with smoking being the most important and preventable risk factor.[1-3]

Characterised by increasingly frequent exacerbations that can be severe, COPD is a significant worldwide cause of morbidity and mortality, with a poor long-term prognosis following hospitalisation for exacerbation (≈50% 5-year mortality).[4] WHO predicts that COPD will be the third leading cause of death worldwide by 2030.[5]

In New Zealand, it is estimated that COPD affects one in seven individuals over the age of 40 and approximately 30,000 New Zealanders are believed to be living with severe COPD requiring stays in hospital. COPD is the fourth leading cause of death in NZ behind cancer, heart disease and stroke.[6] In 2013, New Zealand was recorded as having the third highest hospitalisation rate for COPD in the OECD, with hospitalisation rates 5.1-fold higher and mortality 2.7-fold higher in the most deprived NZDep quintile than in the least deprived.[7-10]

Māori and Pacific people in New Zealand are disproportionately affected by COPD.[11] Māori exhibit a prevalence of COPD over twice that of non-Māori, partly due to the higher rate of smoking in this population.[11] Māori COPD hospitalisation rates are high at over three times that of non-Māori, with Māori

having over twice the rate for mortality.[9] Importantly, Māori are as much as 10 years younger at the time of first hospitalisation for their COPD.[12] Furthermore, amongst New Zealanders aged 50-64 years of age, Māori are five-fold more likely to die from COPD-related causes than non-Māori, with the disease tending to present in Māori up to 20 years earlier than non-Māori.[11]

Diagnosing and assessing COPD

Of the estimated 200,000 adults in New Zealand with COPD, only 1 in 4 to 5 will have had the diagnosis confirmed.[8] By the time COPD is diagnosed, nearly 50% of lung function may already be lost.[4] Any adult over 40 years of age presenting with dyspnoea, chronic cough or sputum production, lower respiratory tract infection, and/or a history of exposure to risk factors for the disease should be assessed for COPD.[1] Risk factors include exposure to tobacco smoke, occupational dusts, fumes and gases, and a strong family history of COPD.[1,2]

Spirometry

Spirometry is required to make the diagnosis of COPD, with the presence of a post-bronchodilator FEV_1/FVC <0.70 confirming the presence of persistent airflow limitation.[1] Furthermore, the GOLD system assesses the severity of airflow limitation, classifying patients into 1 of 4 categories based on post-bronchodilator FEV_1 results:[4]

GOLD 1 (Mild; FEV_1 ≥80% predicted)

GOLD 2 (Moderate; 50% ≤FEV_1 <80% predicted)

GOLD 3 (Severe; 30% ≤FEV_1 <50% predicted)

GOLD 4 (Very severe: FEV_1 <30% predicted).

Spirometry results in conjunction with patient symptoms and exacerbation history are vital for the diagnosis. FEV_1 is an unreliable marker of an individual's severity level of breathlessness, exercise limitation and health status impairment.[1,13] For this reason, the classification of airflow limitation is no longer included in the GOLD ABCD treatment classification grid (**Figure 1**) used to guide pharmacologic treatment, which is now based primarily on symptoms and exacerbation history, emphasising the importance of these two factors in guiding therapies in COPD.[1]

2017 GOLD COPD assessment tool

The goals of COPD assessment are to confirm the diagnosis, to establish the level of airflow limitation, the impact of symptoms on the patient's health status and quality of life as well as the risk of future exacerbations, hospital admissions or death. These factors are combined in the new 2017 GOLD COPD assessment tool designed to guide treatment (**Figure 1**).[1] The tool incorporates the COPD Assessment Test (CAT; http://www.catestonline.org; **Figure 2**), which determines the impact of COPD symptoms on wellbeing and daily life, and assesses response to treatment, and the Modified Medical Research Council (mMRC) Dyspnoea Scale (**Table 1**) for quantifying the degree of breathlessness.[2]

Patients are classified in the grid as either Group A (low level symptoms and low risk of exacerbation), Group B (high level of symptoms and low risk of exacerbation), Group C (low level of symptoms and high risk of exacerbation, or Group D (high level of symptoms and high risk of exacerbation).[1]

Figure 1. The refined 2017 GOLD combined assessment of COPD tool.[1]

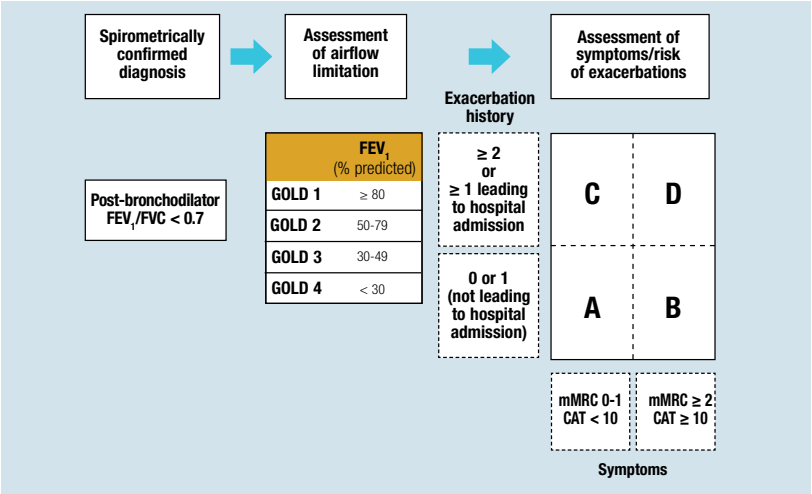


Figure 2: CAT COPD Assessment Test (http://www.catestonline.org)

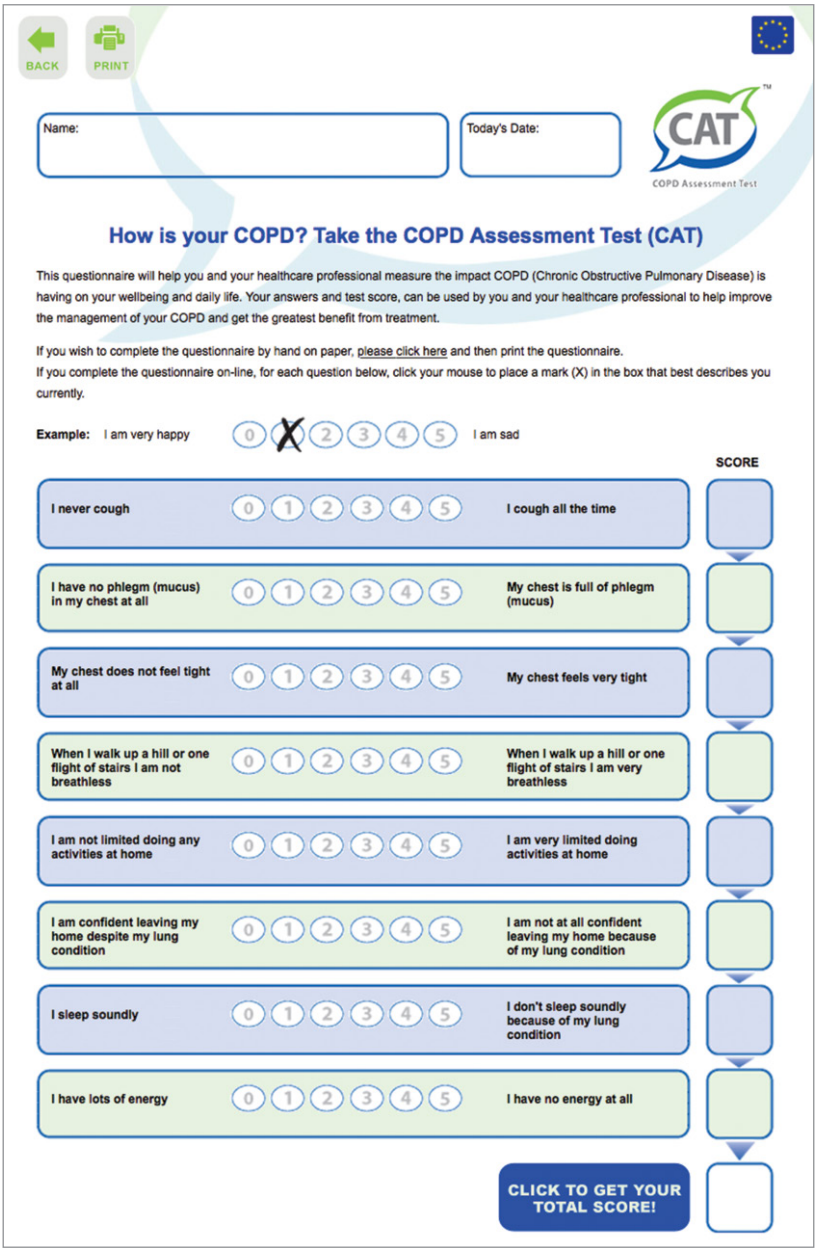


Table 1. Modified Medical Research Council (mMRC) Dyspnoea scale for grading the severity of breathlessness during daily activities[2]

Grade	Symptom complex
0	I only get breathless with strenuous exercise
1	I get short of breath when hurrying on level ground or walking up a slight hill
2	On level ground, I walk slower than people of the same age because of breathlessness, or I have to stop for breath when walking at my own pace on the level
3	I stop for breath after walking about 100 metres or after a few minutes on level ground
4	I am too breathless to leave the house or I am breathless when dressing or undressing

Non-pharmacologic interventions

Smoking cessation is the single most important intervention (and the only one proven to improve disease outcome) and must be actively encouraged in patients with COPD.

Education and self-management is an important component of care in COPD patients, and should be actively encouraged and supported by healthcare professionals.[1] The aim is to motivate, engage and coach the patient to develop skills to adapt their health behaviours and optimally manage their disease. Based on the GOLD groups, a personalised self-management education plan could include:[1]

Groups A, B, C and D: smoking cessation, addressing other behavioural risk factors, regular physical activity, adequate sleep and a healthy diet

Groups B and D: learning energy conservation techniques, learning to manage breathlessness and stress management strategies

Groups C and D: monitoring and managing worsening symptoms, avoiding aggravating factors, having a written action plan and maintaining regular contact/communication with a healthcare professional

Group D: palliative strategies and advanced care directives.

Specific non-pharmacologic interventions to prevent and manage COPD include the following:

Identification and reduction of exposure to risk factors such as tobacco smoke, indoor and outdoor air pollution, and occupational exposures[1]

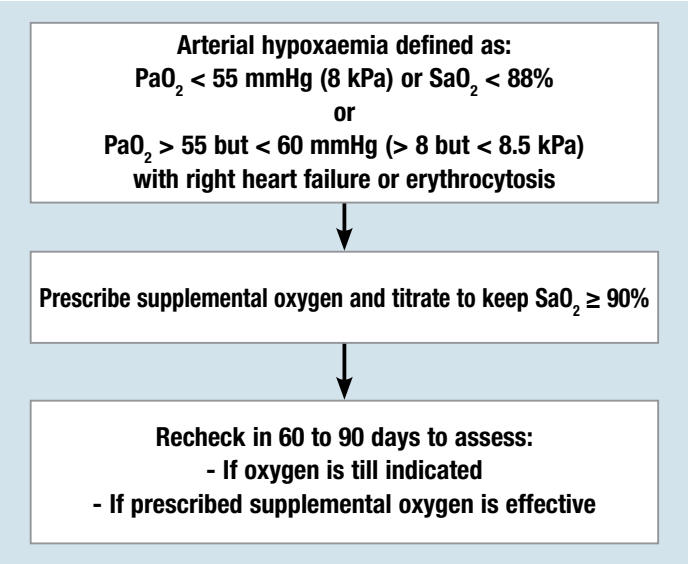
Influenza and pneumococcal vaccination has proven efficacy in COPD patients and should be undertaken as per the Immunisation Handbook[14-17]

Pulmonary rehabilitation, an exercise and education program that aims to improve functional exercise capacity and quality of life, can reduce hospitalisations and when initiated early significantly reduces rates of re-hospitalisation in COPD.[4] It is critical that rehabilitation be introduced early, before the patient is debilitated, and there is strong evidence that such intervention is very effective.[18] All patients with a diagnosis of COPD should have the opportunity to participate in a pulmonary rehabilitation programme, particularly patients in GOLD grade B, C and D.[1] In areas where such a programme is not available, referral to a physiotherapist or exercise physiologist may be appropriate. Home-based pulmonary rehabilitation may be beneficial.[19]

Exercise and diet play a role in COPD with physical activity, weight management and nutritional advice being extremely important factors in the management of this disease.[11,20,21] A diet rich in fruit, vegetables and fibre has been shown to be beneficial in COPD.[22]

Oxygen therapy is indicated long-term for some patients.[1] The following algorithm (**Figure 3**) is a useful resource for the prescription of supplemental oxygen to COPD patients.[1]

Figure 3. Algorithm for the prescription of supplemental oxygen to COPD patients.[1]



Pharmacologic treatment of stable COPD

To date, no inhaler-based pharmacotherapy has been proven to prevent disease progression or reduce mortality in COPD.[4]

The goals of pharmacologic treatment in COPD are to:[4]

- relieve symptoms
- improve exercise tolerance
- improve health status
- reduce the risk and severity of exacerbations (especially hospitalisations)
- prevent disease progression and reduce mortality
- have an acceptable safety profile with minimal side effects
- prevent complications of non-COPD comorbidities especially those related to cardiovascular morbidity and mortality (e.g. statin therapy, for which there are numerous observational studies and three small RCTs indicating benefit in COPD patients).[23-26]

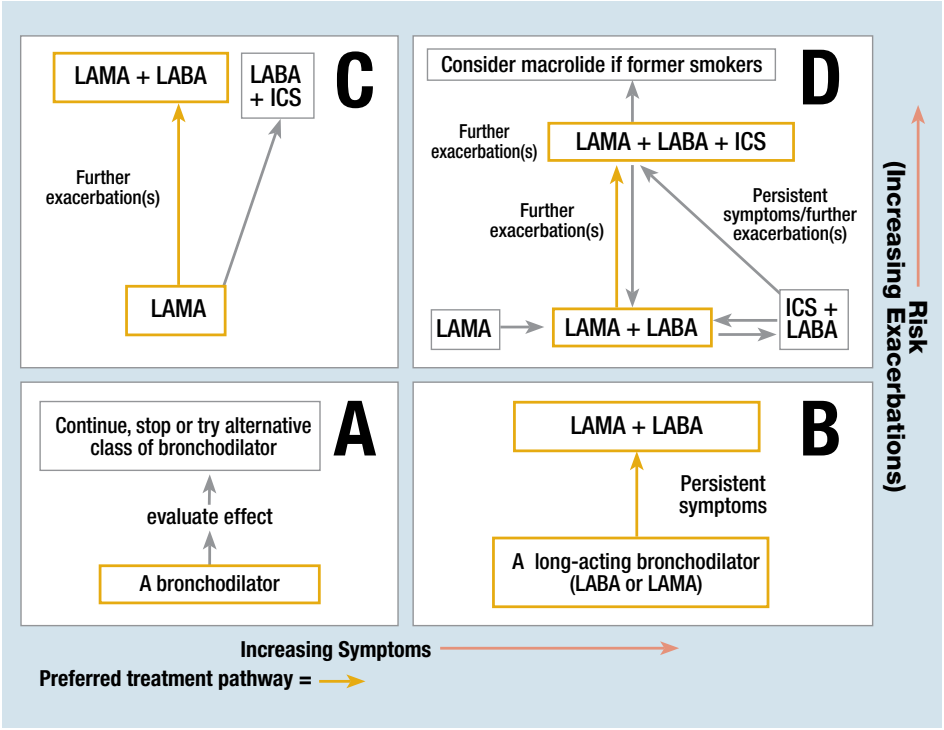
The GOLD initiative proposes a model for inhaler-based management of COPD according to the individualised assessment of symptoms and exacerbation risk (**Figure 4**).[1] The model includes both escalation and de-escalation strategies and highlights preferred treatment pathways. The subsidised inhaled medications available for the treatment of stable COPD in New Zealand are listed in **Table 2 and Figure 5**.

Table 2. Subsidised inhaled medications for the treatment of stable COPD in New Zealand.[27-30] The most recent medicines to be funded are highlighted in bold

	Agent	Trade name	Dose	Frequency	Subsidised inhaler device	Subsidy
SABAs	Salbutamol	Respigen; SalAir; Ventolin	100-200 mcg (1 to 2 puffs of 100 mcg) Max dose: 200 mcg, four times daily	As needed, up to four times daily	MDI – spacer recommended	Fully subsidised without restriction except for Ventolin, which is partially subsidised
	Terbutaline	Bricanyl Turbuhaler	250-500 mcg (1 to 2 inhalations of 250 mcg) Max single dose: 1.5 mg	As needed	DPI breath-activated device	Fully subsidised
SAMAs	Ipratropium bromide	Atrovent	40 mcg Max single dose: 80 mcg	Four times daily	MDI – spacer recommended	Fully subsidised
SAMA plus SABA combination	Ipratropium bromide + salbutamol	Duolin HFA	20 + 100 mcg Two puffs Max dose: 12 puffs in 24 hrs	Four times daily	MDI – spacer recommended	Fully subsidised
LABAs	Formoterol fumarate	Foradil; Oxis Turbuhaler	12-24 mcg Max single dose: up to 24 mcg	Once or twice daily	DPI breath-activated device; breath-activated device with each dose in a capsule	Partially subsidised
	Indacaterol	Onbrez Breezhaler	150-300 mcg Max dose: 300 mcg daily	Once daily	DPI breath-activated device with each dose contained in a capsule	Fully subsidised
	Salmeterol	Serevent Accuhaler	25-50 mcg (1 to 2 puffs of 25 mcg) Max dose: 50 mcg, twice daily	Twice daily	DPI breath-activated device with each dose contained in a disc of eight doses	Fully subsidised
		Meterol; Serevent	25-50 mcg (1 to 2 puffs of 25 mcg) Max dose: 50 mcg, twice daily	Twice daily	MDI	Fully subsidised
LAMAs	Glycopyrronium	Seebri Breezhaler	Powder: 50 mcg	Once daily	DPI breath-activated device with each dose contained in a capsule	Fully subsidised with written endorsement
	Tiotropium	Spiriva HandiHaler	Powder: 18 mcg	Once daily	DPI breath-activated device with each dose contained in a capsule	Fully subsidised with Special Authority
		Spiriva Respimat	**Solution: 2.5 mcg Two puffs once daily at the same time each day**	**Once daily**	**MDI: Solution in a cartridge delivered as a soft mist**	**Fully subsidised with Special Authority**
	Umeclidinium	**Incruse Ellipta**	**62.5 mcg One inhalation once daily**	**Once daily**	**DPI breath-activated device**	**Fully subsidised with written endorsement**
LAMA plus LABA combinations	**Glycopyrronium + indacaterol**	**Ultibro Breezhaler**	**50 + 110 mcg One puff once daily**	**Once daily**	**DPI breath-activated device**	**Fully subsidised with Special Authority**
	Tiotropium + olodaterol	**Spiolto Respimat**	**2.5 mcg + 2.5 mcg Two puffs *once* daily at the same time each day**	**Once daily**	**MDI Solution in a cartridge delivered as a soft mist**	**Fully subsidised with Special Authority**
	Umeclidinium + vilanterol	**Anoro Ellipta**	**62.5 + 25 mcg One inhalation at the same time each day**	**Once daily**	**DPI breath-activated device**	**Fully subsidised with Special Authority**
Corticosteroids	Beclomethasone	Beclazone; Qvar	Max dose: 1000 mcg per day	Twice daily	MDI	Fully subsidised
	Budesonide	Pulmicort	100-800 mcg	Twice daily	DPI	Fully subsidised
	Fluticasone propionate	Flixotide; Flixotide Accuhaler; Floair	100-500 mcg Max dose: 1 mg twice daily	Twice daily	MDI, DPI	Fully subsidised
Corticosteroid plus LABA combinations	Budesonide + formoterol	Symbicort Turbuhaler	200 + 6 mcg Two puffs Max dose: 4 puffs daily	Twice daily	DPI breath-activated device	Fully subsidised
			400 + 12 mcg One puff Max dose: 2 puffs daily	Once daily	DPI breath-activated device	Fully subsidised
		Vannair	200 + 6 mcg Two puffs Max dose: 4 puffs daily	Twice daily	MDI	Fully subsidised
	Fluticasone propionate + salmeterol	Seretide; Rexair	125 + 25 mcg Two puffs	Twice daily	MDI – spacer recommended	Fully subsidised
		Seretide Accuhaler	250 + 50 mcg One puff Max dose: 500 + 50 mcg twice daily	Twice daily	DPI breath-activated [Accuhaler]	
	Fluticasone furoate + vilanterol	**Breo Ellipta**	**100 + 25 mcg One inhalation**	**Once daily**	**DPI breath-activated**	**Fully subsidised**

SABA = short-acting β_2-agonist; SAMA = short-acting muscarinic antagonist; LABA = long-acting β_2-agonist; LAMA = long-acting muscarinic antagonist; mcg = microgram; max = maximum; MDI = metered dose inhaler; DPI = dry powder inhaler; mg = milligram.

Figure 4. Pharmacologic treatment algorithm for COPD by GOLD group. Highlighted boxes and arrows depict preferred treatment pathways.[1]



ICS = inhaled corticosteroid; LABA = long-acting β-agonist; LAMA = long-acting muscarinic antagonist

The new inhaler-based treatment recommendations according to GOLD group are further detailed as follows:[1]– refer **Figure 4**

Group A: All group A patients should be offered a bronchodilator based on it's effects on breathlessness; options are SABA and/or SAMA or LAMA or LABA.

Group B: Initial therapy should consist of LAMA or LABA progressing to LAMA/LABA if patient has persistent symptoms. If the addition of a second bronchodilator does not improve symptoms, step down treatment again to a single bronchodilator. Consider any comorbidities that may be contributing to the symptomatology.

Group C*: Initial therapy should consist of a single LAMA, progressing to LAMA/LABA (preferred) or ICS/LABA (alternative) if patient has persistent exacerbations.

Group D*: Initial therapy should consist of LAMA/LABA (preferred) or ICS/LABA in asthma-COPD overlap syndrome** (ACOS) patients or those with high blood eosinophil counts. If patient develops further exacerbations, triple therapy with LAMA/LABA/ICS is recommended. If the patient still develops further exacerbations, stopping ICS or trial of macrolide could be considered, preferably following specialist referral.

*There is limited evidence supporting the treatment recommendations for groups C and D – Trilogy[38] and Trinity[37] RCTs showing superiority of triple therapy over LABA/ICS in regards to reducing exacerbations.

**Asthma and COPD may be present concurrently in some patients. These patients tend to experience more frequent exacerbations, more rapidly declining lung function and reduced quality of life than those with COPD alone.[4]

Inhaled corticosteroids

The majority of studies have found that regular use of ICSs alone has no modifying effect on the long-term decline of FEV_1 in COPD patients.[32] Further meta-analyses assessing effects of ICS use alone on mortality in COPD patients are inconclusive.[32] Therefore ICSs are not recommended for monotherapy in COPD. Some nervousness exists regarding the use of ICSs in combination inhalers in light of a possibility of an increased risk of pneumonia and other respiratory comorbidities in some patients.[32-35] The risk/benefit of corticosteroids in COPD needs to be considered on a case-by-case basis.[27] There is evidence that ICSs combined with LABAs are more effective in patients who experience frequent exacerbations where chronic bronchitis predominates and in those with a mixed ACOS phenotype.[36]

Studies investigating the withdrawal of ICSs in COPD have demonstrated equivocal findings regarding the impact on lung function, symptoms and exacerbations; these differences may be due to differences in study methodology including the use of long-acting bronchodilators, which may minimise the effect of ICSs withdrawal.[1] No evidence of harm from withholding ICS treatment has been shown so far for GOLD group 'D' and could be trialled in those more prone to pneumonia on a case-by-case basis.[1] *Post hoc* analysis suggests that patients with low blood eosinophil count (<150/μL) are likely to be steroid resistant while those with >300/μL are likely to be steroid responsive. There is some evidence that ICSs as part of triple therapy in late stage COPD for those who frequently exacerbate may be helpful (see TRINITY[37] and TRILOGY[38] studies) but should be based on individual patient need due to the concerns around possible increased incidence of pneumonia, although it has been suggested that this is a class and dose effect.

Managing exacerbations

The definition of an exacerbation of COPD is an acute worsening of respiratory symptoms mainly dyspnoea resulting in additional therapy.[1] Exacerbations are complex events that involve an increase in airway inflammation, mucous production and marked gas trapping. Individual patient characteristics will influence how the event is perceived by the individual and therefore what level of treatment is sought. Most exacerbations are self-limiting but symptoms may last up to 7-10 days. Exacerbation grade and management are referred to as either –

- Mild where an increased use of short-acting bronchodilator (SABD) only is needed. Many of these are self-managed and are not reported to a healthcare provider.
- Moderate, requiring an increased use of SABD +/- antibiotics or oral corticosteroids
- Severe, necessitating an emergency department visit with or without hospital admission. These severe exacerbations might be associated with acute respiratory failure.

Many patients with COPD have a complex health picture with multi-morbidity so it is important to distinguish a COPD exacerbation from an acute event of another cause, namely, an acute coronary event, worsening heart failure, pulmonary embolism, lung cancer

or pneumonia.[1] Prompt and appropriate treatment of exacerbations is of utmost importance as is assessing response to therapy.[1] The goal of such treatment is to gain rapid control of presenting symptoms and prevent the development of complications such as pneumonia, heart failure and acute coronary syndrome. Failure of resolution or improvement of presenting symptoms over 3-4 weeks might prompt consideration of chest Xray to identify alternate diagnoses including lung cancer. Most exacerbations are managed in the outpatient setting. The GOLD strategy key points for the management of exacerbations are as follows:[1]

- SABAs and/or SAMAs are recommended as initial bronchodilators for an acute exacerbation*
- Systemic corticosteroids can improve FEV_1, oxygenation and shorten recovery and hospitalisation duration, but should not be administered for >5-7 days
- Antibiotics, if indicated, can be beneficial, but should not be administered for >5-7 days
- Methylxanthines are not recommended
- Oxygen therapy is a key component of hospital treatment for an exacerbation
- Non-invasive mechanical ventilation should be the first mode of ventilation used in those with acute respiratory failure
- Initiation of adjunct therapy appropriate to exacerbation severity, such as fluid management, treatment of comorbidities, nutritional support and establishing a comprehensive plan to manage future events
- After hospitalisation pulmonary rehabilitation should be considered.

*It is recommended that patients use an MDI one puff every hour for two or three doses and then every 2-4 hours as needed, rather than continuous nebulisation. It is also recommended that if the patient is receiving a LAMA, LABA and/or ICS they continue to receive these agents throughout the exacerbation or restart them as soon as possible before hospital discharge.

Figure 5: Inhaler devices funded for COPD in New Zealand [Adapted from Canterbury Clinical Network][31]

SABA = short-acting β_2-agonist; SAMA = short-acting muscarinic antagonist; LABA = long-acting β_2-agonist; LAMA = long-acting muscarinic antagonist.

Importance of correct inhaler technique

Correct inhaler technique is extremely important in treating COPD and it is imperative that patients understand the actions and treatment aims of their various inhalers including how to use their inhaler/s most effectively.[1] Patients should be taught the correct technique of their particular inhalers prior to leaving the clinic and are encouraged to thoroughly read the patient information leaflet provided with the device.[1] Inhaler technique should be assessed before concluding that the current therapy is considered ineffective.

Evidence suggests that on average over two-thirds of patients with COPD make at least one error when using an inhalation device and not surprisingly there is a significant relationship between poor inhaler use and symptom control.[39-41] Patients using multiple devices (particularly with different delivery mechanisms), those who are older, and those who lack previous education on inhaler use are more likely to exhibit poor technique.[42] The most common errors in delivery device use involve inhalation duration, coordination, exhalation manoeuvre before inhalation and breath-holding following dose inhalation.[1] Patients should be encouraged to bring their own inhalers to the clinic for assessment and regular review of inhaler technique where appropriate.[1] Several videos demonstrating correct inhaler technique are available here.

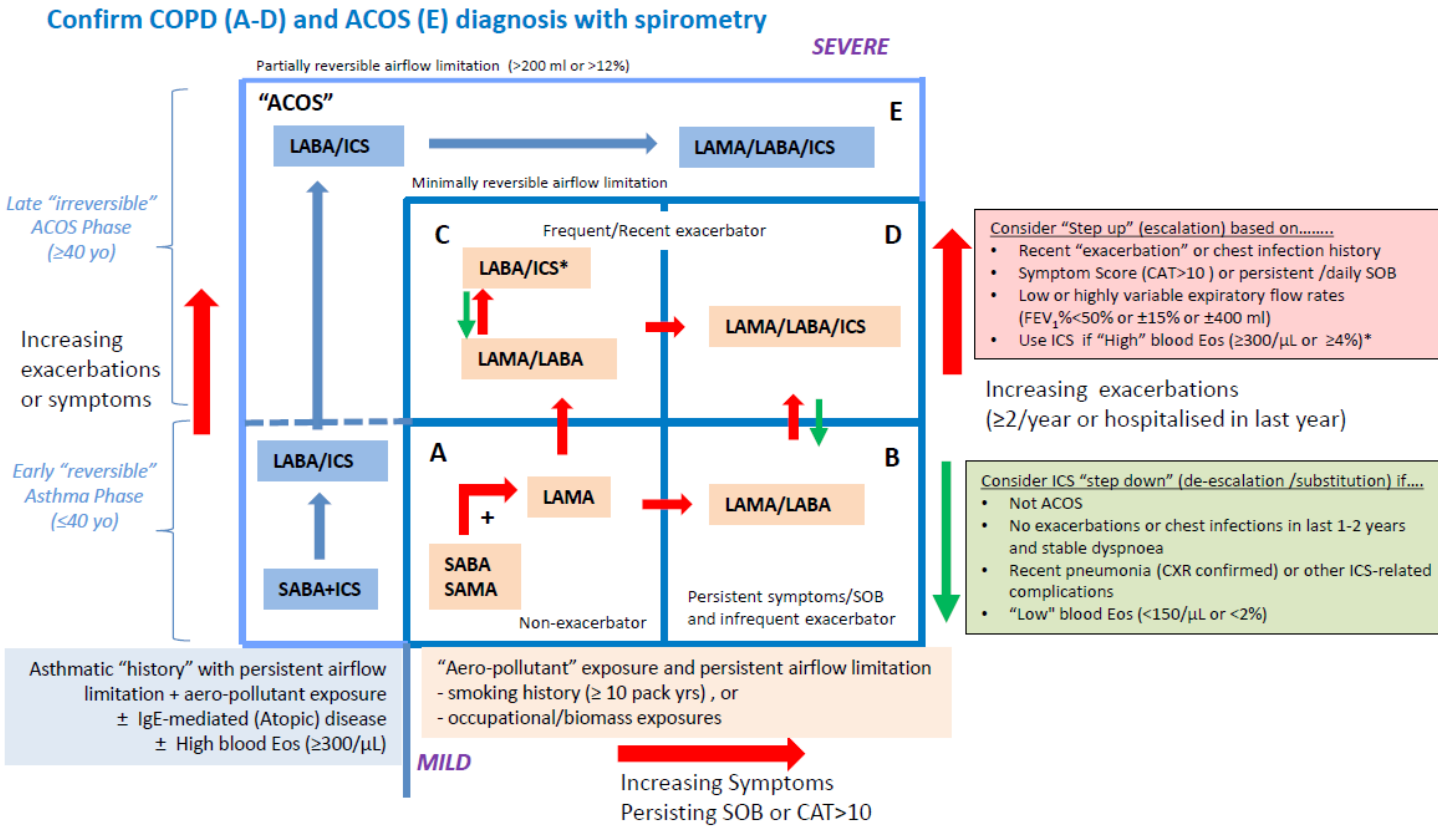
Ongoing care for patients with COPD

- It is essential that patients be educated to identify and treat COPD exacerbations early, as a delay of more than 24 hours doubles the likelihood of hospital admission[2]
- All patients with COPD should have a written management/action plan detailing which bronchodilator to use and when to increase it, when to start antibiotics and corticosteroids and when to seek medical help
- Inhalation device and spacer usage technique should be checked at each visit
- Ideally reassess COPD patients at least annually.[2] At each clinic visit an assessment should include current symptom severity (CAT score) and risk of exacerbation. Spirometry should be repeated on a case-by-case basis according to reported exercise tolerance. Patients may benefit from patient support groups.

Co-morbidity and multi-morbidity

COPD is now recognised as a multi system disease characterised by premature aging and several comorbid diseases including; coronary artery disease, heart failure, sleep apnoea, gastro-oesophageal reflux, depression/anxiety, osteoporosis and lung cancer.[1] Recent attention has turned to the fact that COPD is not just underdiagnosed but is undertreated, particularly with regards to these comorbidities.

Figure 6. Alternate schema to the pharmacologic treatment algorithm for COPD by GOLD group, adapted for the New Zealand setting.[43]



ACOS = asthma-COPD overlap syndrome; CAT = COPD-assessment test; CXR = chest radiograph; FEV1 = forced expiratory volume in 1 sec; ICS = inhaled corticosteroids; LABA = long-acting β-agonist; LAMA = long-acting muscarinic antagonist; SABA = short-acting β-agonist; SAMA = short-acting muscarinic antagonist; SOB = shortness of breath

EXPERT'S CONCLUDING REMARKS:

While the GOLD 2017 recommendations are for the most part supported by clinical trial data, gaps in the evidence justifying some of these recommendations remain. The de-emphasis on spirometry in the 2017 strategy should not discourage the routine use of quality spirometry in the primary care setting. The above recommendations should be considered as a framework from which to initiate and escalate therapy in the management of COPD patients.

In New Zealand, the choice of inhalers is directed to some degree by PHARMAC requirements. In order to comply with these requirements, we have modified the GOLD 2017 criteria to suit a New Zealand specific setting as set out in the schema (**Figure 6**).

REFERENCES:

1. Global Initiative for Chronic Obstructive Lung Disease. 2017 Report. Available from: http://www.goldcopd.org (Accessed May 2017)
2. The COPD-X Plan. Available from: http://copdx.org.au/copd-x-plan/ (Accessed May 2017)
3. van der Molen and Schokker S. *Proceedings of the American Thoracic Society*, Vol. 6, No. 8 (2009), pp. 704-06
4. Global Initiative for Chronic Obstructive Lung Disease (GOLD). 2016. Available from: http://goldcopd.org (Accessed May 2017)
5. MOH. Mortality and Demographic Data 2009. Wellington 2014; Available from: http://www.health.govt.nz/publication/mortality-and-demographic-data-2009 (Accessed May 201
6. World Health Organisation. World Health Statistics 2008. Available from: http://www.who.int/whosis/whostat/EN_WHS08_Full.pdf?ua=1 (Accessed May 2017)
7. Shirtcliffe P et al. *Eur Respir J* 2007;30:232-9
8. Town I et al. 2003. Available from: http://asthmafoundation.org.nz/wp-content/uploads/2012/03/burdenCOPD.pdf (Accessed March 2016)
9. Telfar-Barnard L et al. 2014 update. Published: 4 April 2015. Available from: https://www.asthmafoundation.org.nz/research/the-impact-of-respiratory-disease-in-new-zealand-2014-update (Accessed May 2017)
10. OECD (2013), Health at a Glance 2013: OECD Indicators, OECD Publishing. Available from: http://dx.doi.org/10.1787/ health_glance-2013-en (Accessed May 2017)
11. Bpac. Available from: http://www.bpac.org.nz/BPJ/2015/February/copd-part1.aspx (Accessed May 2017)
12. Hopkins RJ et al. *Am J Respir Crit Care Med* 193;2016:A6658.
13. Augusti A et al. *Respiratory Research* 2010;11:122
14. Nichol KL et al. *N Eng J Med.* 1994;331:778-84
15. Poole PJ, et al. *Cochrane Database Syst Rev.* 2006;(1):CD002733
16. Furumoto A et al. *Vacccine* 2008;26(33):4284-9
17. Ministry of Health. 2017. Immunisation Handbook 2017. Wellington: Ministry of Health. Available from: http://www.health.govt.nz/publication/immunisation-handbook-2017 (Accessed May 2017)
18. Hanekom S et al. *Clin Rehabil.* 2011;25(9):771-87
19. Holland AE, et al. ATS International Conference 2016; Abstract 2620. Available from: http://www.atsjournals.org/doi/abs/10.1164/ajrccm-conference.2016.193.1_ MeetingAbstracts.A2620
20. Garcia-Aymerich J et al. *Thorax* 2006;61(9):772-8
21. Garcia-Aymerich J et al. *Chest* 2009;136(1):62-70
22. Varrasso R and Shaheen SO. *Thorax* 2017;72(6):491-92
23. Morz RM et al. *J Physiology and Pharmacology* 2015;66:111-128
24. Maneechotesuwan K et al. *Chest* 2015; 148:1164-76
25. Lee TM et al. *Am J Cardiol.* 2008; 101:530-5
26. Ingebrigtsen TS et al. *Thorax* 2015;70:33-40
27. Bpac NZ. Available from: http://www.bpac.org.nz/BPJ/2015/February/copd-part2.aspx (Accessed May 2017)
28. Pharmaceutical Management Agency Update. New Zealand Pharmaceutical Schedule. Effective March 2016
29. Medsafe data sheets. Available from: http://www.medsafe.govt.nz (Accessed May 2017)
30. NZ formulary. Available from: http://nzf.org.nz (Accessed May 2017)
31. Canterbury Clinical Network. Available from: http://www.healthinfo.org.nz/patientinfo/Funded%20Inhalers%20in%20NZ%20CCN.pdf (Accessed May 2017)
32. Yang IA et al. *Cochrane Database Syst Rev.* 2012;7:CD002991
33. Cazzola M and Page C. L *Breathe* 2014; DOI: 10.1183/20734735.014813
34. Dong Y-H et al. *Chest J.* 2014;145:1286
35. Andréjak C et al. *Thorax* 2013;68:256-62
36. Miravitlles M et al. *Eur Respir J* 2013;41: 1252-56
37. Vestbro J et al. *The Lancet* 2017; 389:1919-29.
38. Singh D et al. Lancet 2016;388(10048):963-73
39. Souza ML et al. *J Bras Pneumol.* 2009;35(9):824-31
40. Melani AS et al. *Respir Med.* 2011;105(6):930-8
41. Sanchis J *Chest* 2016;150:394
42. Rootmensen GN et al. *J Aerosol Med Pulm Drug Deliv.* 2010;23(5):323-8
43. Young RP and Hopkins RJ. *Eur Respir J.* 2017;49(1):pii: 1601970

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