

Research Review™

EDUCATIONAL SERIES

Managing chronic obstructive pulmonary disease (COPD) in general practice

About the Experts



**Professor
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Professor Lutz Beckert is the Head of Department of Medicine of the University of Otago, Christchurch. He is also a Respiratory Physician at Canterbury District Health Board with particular clinical interests in interstitial lung disease, pulmonary vascular disease, respiratory physiology and COPD (chronic obstructive pulmonary disease). Lutz is happy to be contacted to discuss research ideas either as a sounding board or considering future collaborations.



**Associate Professor
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Jim Reid graduated in medicine at the University of Otago Medical School in Dunedin New Zealand. He had previously trained as a pharmacist. He undertook his postgraduate work at the University of Miami in Florida. Currently he is Deputy Dean of the Dunedin School of Medicine. He has a private family medicine practice at the Caversham Medical Centre, Dunedin, New Zealand. He is a Distinguished Fellow of the Royal New Zealand College of General Practitioners and is also a Fellow of the American College of Chest Physicians. He has a special interest in Respiratory Medicine and has published widely in Asthma and COPD. He is active in research in respiratory medicine and has had wide international lecturing experience.

Abbreviations used in this review

COPD = chronic obstructive pulmonary disease

FEV₁ = forced expiratory volume in one second

FVC = forced vital capacity

LABA = long-acting β_2 -agonist

LAMA = long-acting muscarinic antagonist

SABA = short-acting β_2 -agonist

SAMA = short-acting muscarinic antagonist

WHO = World Health Organisation

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This publication is intended as an educational resource for health professionals. It discusses the diagnosis and management of chronic obstructive pulmonary disease (COPD) and presents the full range of treatments available for this condition in New Zealand. It is intended to help readers stay informed of developments and advancing clinical practice in this area.

As of 1 March 2016, PHARMAC has funded six new medicines, improving treatment options for patients with COPD.¹ The newly funded medicines for inhalation are:

- The long-acting muscarinic antagonists (LAMAs) **tiotropium bromide solution [Spiriva Respimat®]** and **umeclidinium powder [Incruse Ellipta®]**
- LAMA plus long-acting β_2 -agonist (LABA) combinations of **tiotropium bromide + olodaterol solution [Spiolto Respimat®]**, **umeclidinium + vilanterol powder [Anoro Ellipta®]** and **indacaterol + glycopyrronium powder [Ultibro Breezhaler®]**
- The inhaled corticosteroid (ICS) plus LABA combination of **fluticasone furoate + vilanterol powder [Breo Ellipta®]**

Glycopyrronium powder for inhalation [Seebri Breezhaler®] and **umeclidinium powder for inhalation [Incruse Ellipta®]** do not require Special Authority but require endorsement from the prescriber that the patient has been diagnosed with COPD using spirometry.

Other relatively recently funded agents include:

- A 800 mL spacer device [**Volumatic**]
- Generic formulations of **salbutamol (a short-acting β_2 -agonist [SABA])**, **fluticasone propionate**, and the LABA/ICS combination of **salmeterol + fluticasone propionate**

NB: Fluticasone furoate and fluticasone propionate are completely different agents with distinct properties.

What is COPD?

Chronic obstructive pulmonary disease (COPD) is a common, chronic respiratory disease that is caused by inflammation leading to a variable combination of emphysema (parenchymal tissue destruction) and obstructive bronchiolitis (small airways disease).² COPD is characterised by airflow limitation that is not fully reversible. This limitation is usually progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases.²

Whilst the most common symptoms of COPD (dyspnoea, cough and increased sputum production) are respiratory in nature, a number of extrapulmonary symptoms (including weight loss, skeletal muscle atrophy and anaemia) and comorbidities (including infections, lung cancer, osteoporosis, cardiovascular disease and depression) may be present.^{2,3}

Who is at risk?

Smoking is the most important and preventable risk factor for COPD.^{4,5} Other risk factors include long-term cannabis use, air pollution, occupational exposure (e.g. to cadmium, asbestos, dusts or silica) and genetic predisposition (including alpha-1 antitrypsin deficiency).⁶

The burden of COPD

COPD is a chronic illness with frequent exacerbations that can be severe; the disease is a significant worldwide cause of morbidity and mortality.³ The long-term prognosis following hospitalisation for COPD exacerbation is poor, with a 5-year mortality rate of approximately 50%.²

WHO predicts that COPD will be the third leading cause of death worldwide by 2030.⁷ Approximately 30,000 New Zealanders are estimated to be living with severe COPD requiring stays in hospital;⁸ 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 in the most deprived NZDep quintile than in the least deprived, and mortality rates that were 2.7-fold higher.^{8,9} Among New Zealanders aged ≥ 65 years COPD was the third leading cause of death in 2012 for Māori and non-Māori females, and the fourth leading cause of death for Māori and non-Māori males.¹⁰

Māori and Pacific people in New Zealand are disproportionately affected by COPD.⁶ Māori exhibit a prevalence of COPD over twice that of non-Māori, partly due to the higher rate of smoking in this population.⁶ Furthermore, amongst New Zealanders aged 50–64 years of age, Māori are 5-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.⁶

DIAGNOSING COPD

Of the estimated 200,000 adults in New Zealand with COPD, only 1 in 4 to 5 will have had the diagnosis confirmed.¹¹ By the time COPD is diagnosed, nearly 50% of lung function may already be lost.²

Assessment of COPD should involve review of the patient's level of symptoms, prediction of future risk of exacerbations, analysis of the severity of the spirometric abnormality and the identification of comorbidities.²

Other illnesses that may present with similar symptoms to COPD and which should be considered in the differential diagnosis include: asthma (see below), respiratory infection, alpha-1 antitrypsin deficiency and tuberculosis.⁶

Breathlessness can be easily quantified using the Modified Medical Research Council Dyspnoea Scale (see **Table 1**).⁴

Table 1: Modified Medical Research Council (mMRC) Dyspnoea scale for grading the severity of breathlessness during daily activities⁴

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

Assessing lung function via spirometry

Spirometry is required to establish a clinical diagnosis of COPD and can be reliably performed in general practice with appropriate training; there are a number of accredited training courses in New Zealand.^{4,12} Peak expiratory flow rate is not useful for assessing COPD. Indications for spirometry include breathlessness, chronic or intermittent cough, frequent or unusual sputum production, relapsing acute infective bronchitis, and risk factors such as exposure to tobacco smoke, occupational dusts and chemicals, and a strong family history of COPD.⁴

The FEV₁ (forced expiratory volume in 1 second) and the FEV₁/FVC (maximum volume of air exhaled forcefully following a maximal inspiration) ratio are reduced in patients with COPD. Patients with airways disease exhale less and slower than patients without airways disease (i.e. the FEV₁/FVC ratio is less than the lower limit of normal – often less than 70%). A post-bronchodilator FEV₁/FVC <0.7 confirms the diagnosis of COPD.²

The volume of air exhaled in the first second compared to a reference population is used to estimate the severity of airflow obstruction (FEV₁% predicted). Patients who exhale less than half of that of people of the same height, age and gender are considered to have severe airflow obstruction.

Information on spirometry training courses in New Zealand may be found at: www.asthmafoundation.org.nz/

Assessing disease severity

The Global Initiative for Chronic Obstructive Lung Disease (GOLD) system assesses the severity of airflow limitation, classifying patients with COPD into 1 of 4 categories, based on post-bronchodilator FEV₁ results.²

GOLD 1: Mild: FEV₁ ≥80% predicted

GOLD 2: Moderate: 50% ≤FEV₁ <80% predicted

GOLD 3: Severe: 30% ≤FEV₁ <50% predicted

GOLD 4: Very severe: FEV₁ <30% predicted

The frequency of exacerbations may increase with the severity of COPD but comorbidities including cardiovascular disease, skeletal muscle dysfunction, osteoporosis, metabolic syndrome, lung cancer, anxiety and depression, peripheral vascular disease and sleep apnoea may be found among all severity groups.¹³

The COPD Assessment Test (CAT: <http://www.catestonline.org>) is useful for determining the impact of COPD symptoms on wellbeing and daily life and for assessing progress with treatment.

Combining results from the CAT and mMRC Questionnaire (**Table 1**) and the GOLD system, the GOLD rubric below was designed to assess risk and improve the management of COPD (**Table 2**).² It takes into consideration symptoms, airflow limitation and exacerbations, as follows:

Symptoms/Breathlessness:

Fewer symptoms (mMRC 0–1 or CAT <10)

patient is (A) or (C)

More symptoms (mMRC ≥2 or CAT ≥10)

patient is (B) or (D)

Airflow limitation:

Low risk (GOLD 1 or 2)

patient is (A) or (B)

High risk (GOLD 3 or 4)

patient is (C) or (D)

Exacerbations:

Low risk: ≤ 1 per year and no hospitalisation for exacerbation

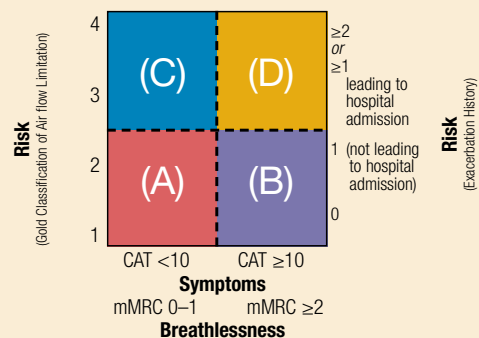
patient is (A) or (B)

High risk: ≥2 per year or ≥1 with hospitalisation

patient is (C) or (D)

Table 2: GOLD combined assessment of COPD²

When assessing risk, choose the **highest risk** according to GOLD grade or exacerbation history. (One or more hospitalisations for COPD exacerbations should be considered high risk).



Patient	Characteristic	Spirometric Classification	Exacerbations per year	CAT	mMRC
A	Low Risk Less Symptoms	GOLD 1-2	≤1	<10	0-1
B	Low Risk More Symptoms	GOLD 1-2	≤1	≥10	≥2
C	High Risk Less Symptoms	GOLD 3-4	≥2	<10	0-1
D	High Risk More Symptoms	GOLD 3-4	≥2	≥10	≥2

Adapted from GOLD Global strategy for the diagnosis, management and prevention of COPD. (2016)

Differentiating COPD from chronic asthma

Asthma and COPD are usually fairly easy to differentiate in the hospital setting but may be less easy to differentiate in general practice. Patient age has a significant impact on the differential diagnosis: in children and young adults, asthma is the most likely chronic airway disease after the exclusion of infection, while in individuals over 35 years of age, COPD is more likely.⁶ Many patients with asthma meet the diagnostic criteria for COPD, reflecting patients with asthma whose airways obstruction is not fully reversible. Pathologically, one of the main differences is that the inflammation in asthma is characterised by the presence of eosinophils and in COPD the inflammation is characterised by the presence of neutrophils; however, in overlap syndrome (see below) there is a mixed inflammatory pattern.²

A new concept of 'treatable traits' in chronic airways diseases has recently been proposed, which would see less emphasis on the need to differentiate between asthma and COPD, and an aim to manage patients with airway disease based on the treatable traits present in each individual.¹⁴ This may well be the way forward in managing such disease.

Asthma-COPD overlap syndrome

Asthma and COPD may be present concurrently in some patients and this condition is referred to as Asthma-COPD overlap syndrome (ACOS). Patients with ACOS tend to experience more frequent exacerbations, more rapidly declining lung function and reduced quality of life than those with COPD alone.² Referral to a respiratory specialist is recommended for these patients.⁶

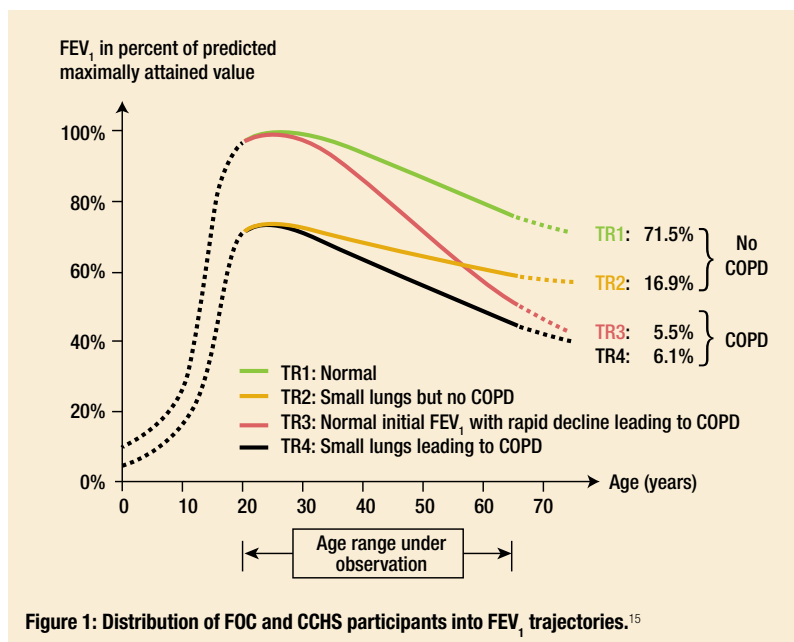
When to consider referral to a respiratory specialist

Australian and New Zealand guidelines for the management of COPD provide helpful guidance on which patients need specialist assessment.⁴ Referral to a specialist respiratory outpatient service may occur at any stage of the disease. Indications for which consultation may be considered are listed in **Table 3**.

Table 3. Indication for considering referral to specialist respiratory outpatient services. Adapted from The COPD-X Plan.⁴

Reason	Purpose
Diagnostic uncertainty and exclusion of asthma	Establish diagnosis and optimise treatment Check degree of reversibility of airflow obstruction
Unusual symptoms such as haemoptysis	Investigate cause including exclusion of malignancy
Rapid decline in FEV ₁ *	Optimise management
Severe COPD	Seek a second opinion
Onset of cor pulmonale	Confirm diagnosis and optimise treatment
Assessment of home oxygen therapy: ambulatory or long-term oxygen therapy	Optimise management, measure blood gases and prescribe oxygen therapy
Bullous lung disease	Confirm diagnosis and refer to medical or surgical units for bullectomy
COPD <40 years of age	Establish diagnosis and exclude alpha-1 antitrypsin deficiency
Assessment for lung transplantation or lung volume reduction surgery	Identify criteria for referral to transplant centres
Frequent chest infections	Rule out co-existing bronchiectasis
Dysfunctional breathing	Establish diagnosis and refer for pharmacological and non-pharmacological management

*Importantly, a rapid decline in FEV₁ is not a prerequisite for COPD. An analysis of data from more than 2500 individuals in Denmark and the USA who participated in the Framingham Offspring Cohort (FOC), the Copenhagen City Heart Study (CCHS) and the Lovelace Smokers Cohort revealed that 52% of those who ultimately developed COPD had a low FEV₁ before the age of 40 years, but a normal decline in FEV₁ thereafter.¹⁵ The other 48% of subjects who developed COPD had a normal FEV₁ before age 40 years and experienced a rapid decline in FEV₁ thereafter, despite similar smoking exposure. **Figure 1** shows the rate of decline in FEV₁ over time among 1622 participants in the FOC and 1242 participants in the CCHS who were aged <40 years at baseline and who underwent spirometry 10 to 27 years after joining their respective cohorts. The subjects were stratified into four trajectories according to lung function (FEV₁ ≥80% or <80% of the predicted value) at cohort inception and the presence or absence of COPD at the last study visit: trajectory 3 depicts those persons who developed COPD with normal baseline FEV₁ and a rapid decline in FEV₁ thereafter, with a mean decline of 53 ml per year; trajectory 4 depicts those who had FEV₁ <80% at baseline, and a significantly smaller subsequent decline in FEV₁ of 27 ml per year.



MANAGING COPD – Non-Pharmacological Interventions

COPD generally should be managed in general practice. However, in some cases, referral to a respiratory service may be appropriate.¹⁶

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.

Pulmonary rehabilitation, an exercise and education programme 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.¹⁷ All patients with a diagnosis of COPD should have the opportunity to participate in a pulmonary rehabilitation programme. 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.¹⁸

Other non-pharmacological interventions such as physical activity, weight management and nutritional advice are extremely important factors in the management of COPD.^{15,19,20} Immunisation against influenza and pneumococcal infection also has proven efficacy in COPD patients and should be undertaken as per the Immunisation Handbook.²¹⁻²⁴

MANAGING COPD – Pharmacological Interventions

Pharmacological management of COPD should be based on an individualised assessment of current symptoms and future risks (see **Table 2**).² The goal of treatment is to relieve symptoms, improve exercise tolerance, improve health status, treat exacerbations, reduce mortality and prevent disease progression, with minimal side effects.² At present, there is no high-quality evidence to suggest that medications can modify the long-term decline in lung function in COPD.^{16,25} Treatment for stable COPD should be initiated according to GOLD class A, B, C or D as discussed in **Table 2**. **Table 4** outlines the suitable treatment options.

Table 4: Treatment options in COPD according to GOLD A, B, C, or D class²

GOLD Class	Recommended first choice	Alternative choice	Other possible treatments
A	SAMA prn or SABA prn	LAMA or LABA or SABA + SAMA	Theophylline
B	LAMA or LABA	LAMA + LABA	SABA +/- SAMA Theophylline
C	ICS + LABA or LAMA	LAMA + LABA or LAMA + PDE-4 inhibitor or LABA + PDE-4 inhibitor	SABA +/- SAMA Theophylline
D	ICS + LABA +/- LAMA	ICS + LABA + LAMA or ICS + LABA + PDE-4 inhibitor or LAMA + LABA or LAMA + PDE-4 inhibitor	N-acetylcysteine* SABA +/- SAMA Theophylline + ICS + LABA + LAMA

SAMA = short-acting muscarinic antagonist; SABA = short-acting β_2 -agonist; LAMA = long-acting muscarinic antagonist; LABA = long-acting β_2 -agonist; ICS = inhaled corticosteroid; PDE-4 = phosphodiesterase-4; prn = as needed.

* N-acetylcysteine is available in New Zealand but is not funded. Prof. Beckert uses N-acetylcysteine about once every 2 years in patients who are highly motivated and who have the main complaint of secretions that they cannot clear. Evidence suggests that long-term use of N-acetylcysteine 600 mg twice daily can prevent exacerbations, especially in moderately-severe disease.²⁶

Bronchodilators

Inhaled bronchodilators are central to the treatment of COPD.²⁷ The existing drug classes, β_2 -agonists and muscarinic receptor antagonists (anticholinergics), increase respiratory skeletal muscle activity and improve ventilator mechanics by relaxing smooth muscle tone.²⁷ The aim of treatment with bronchodilators is to alleviate bronchial obstruction and airflow limitation, as well as air trapping, thus making it easier for patients to breathe.^{27,28} The GOLD guidelines state that the choice of bronchodilator should be based on the availability of the agents, and the patient's symptom relief and side effects.²

Table 5 lists the subsidised inhaled bronchodilators available in New Zealand for patients with stable COPD. The newly funded medicines are depicted in bold. Medicines only partially subsidised are indicated.

Inhaled corticosteroids

There is growing concern that inhaled corticosteroids may increase the risk of pneumonia and other respiratory comorbidities in some patients.^{27,29-31} The risk/benefit of corticosteroids in COPD needs to be weighed up on a case by case basis.^{6,16}

There is evidence that inhaled corticosteroids are more effective in patients who experience frequent exacerbations with chronic bronchitis predominance and in those with overlap between COPD and asthma.³² The subsidised inhaled corticosteroids available in New Zealand for patients with stable COPD are listed in **Table 5**.

Combination inhalers

Combination inhalers allow treatment with the convenience of a single delivery system, which is expected to aid adherence.³³ Combination therapies in COPD have proven efficacy over the individual components.³³ The subsidised combination inhalers available in New Zealand for treating COPD are listed in **Table 5**.

Antibiotics

Reserve the use of antibiotics for treating infections and acute exacerbations of COPD.² In such cases, a 5–10 day course of antibiotics is recommended (suitable antibiotics include amoxicillin 500 mg thrice daily or doxycycline 100 mg twice daily).⁴ Antibiotic prophylaxis or continuous use is not recommended.¹⁶ Sputum culture is not recommended as being necessary, unless the patient does not respond to treatment.

Theophylline

Theophylline is now rarely used in COPD; however, it is a useful agent when there are no other viable options. The agent should be reserved for patients with difficult-to-control symptoms in spite of standard treatment or where inhalation therapy is not suitable.¹⁶ Theophylline is difficult to use; to avoid side effects (principally nausea), a 'start low go slow' principle should apply. It is important to monitor blood levels, especially in patients with renal impairment.

Oral corticosteroids

A 5-day course of oral corticosteroid (e.g. prednisone 40 mg once daily in the morning for moderate to severe exacerbations) has been shown to reduce the severity of COPD exacerbations and improve recovery time.⁴ Long-term corticosteroid therapy should not be used in COPD.⁴ In patients who are not steroid-dependent it is not necessary to taper the steroids in courses of up to 10 days.

A STEPWISE APPROACH TO PHARMACOLOGICAL TREATMENT

Current guidelines recommend a stepwise approach to management based on disease severity (see **Table 4** for an outline of the suitable treatment options for COPD in GOLD class A, B, C, or D).^{2,4} Treatment must be individualised to the patient and care should be taken not to duplicate medicine classes whenever changes to treatment are made.¹⁶ Remember to check device usage technique at each visit.⁴

Managing exacerbations

Prompt treatment of exacerbations is of utmost importance. Good evidence indicates that treatment of an exacerbation within the first 24 hours is associated with a 30% decrease in hospitalisation. Management includes:¹⁶

- Use increased doses of inhaled bronchodilators (e.g. salbutamol 400–800 mcg every 3 to 4 hours). If dosing every 3 hours does not bring relief, advise the patient to seek medical advice
- A 5-day course of oral corticosteroid, e.g. prednisone, if a moderate to severe exacerbation
- A 5-day course of oral antibiotics, but only if there are signs of chest infection
- Increase bronchodilators

Hospital admission

Hospitalisation may be required for patients experiencing an exacerbation who exhibit:⁴

- A significant increase in the intensity of symptoms
- Dyspnoea that is affecting their ability to sleep or eat
- An inability to cope at home
- An inadequate response to community-based treatments
- Features suggestive of respiratory failure, i.e. confusion, drowsiness, restlessness, and cyanosis
- New-onset arrhythmia
- SpO₂ <92% on pulse oximetry if previously higher
- Deteriorating cor pulmonale

Patients exhibiting hypoxaemia during a COPD exacerbation may require controlled oxygen delivered at a rate of 0.5–2.0 L/min.⁴ Following discharge from hospital, it is recommended that the patient be followed-up by the primary care team within 7 days.¹⁶

Advanced COPD

Initiating advanced care planning in patients with COPD is not easy, as most patients have lived with respiratory symptoms all their life; they see themselves living with, and not dying of, COPD.³⁴ New Zealand research suggests that patients identify six recurring transition points, when it may be possible to engage in a discussion about end-of-life and focus on palliating symptoms of anxiety and breathlessness, utilising integrated services offered by allied health, palliative care and respiratory medicine (see **Fig. 2**).³⁵

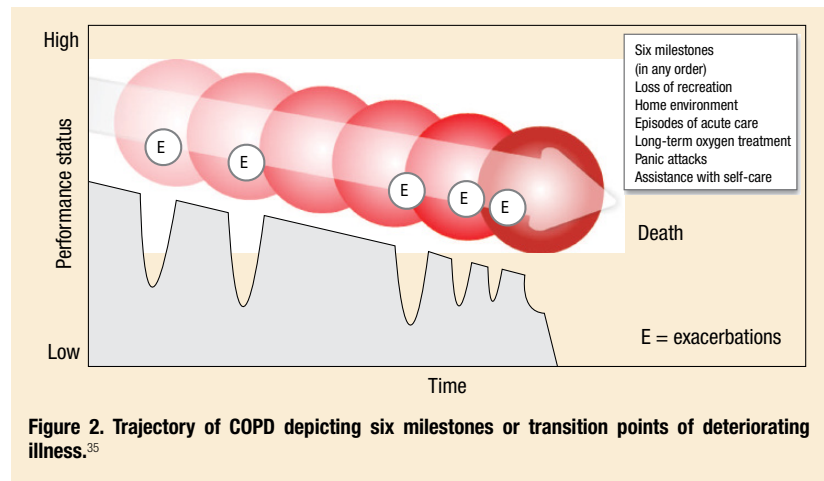


Figure 2. Trajectory of COPD depicting six milestones or transition points of deteriorating illness.³⁵

Ongoing care for patients with COPD

- 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
- 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 admission⁴
- It is also prudent to check inhalation device and spacer usage at each visit, as up to 90% of patients do not use their devices correctly³⁶
- Patients may benefit from patient support groups
- Ideally, COPD patients should be reassessed at least annually.⁴ Assessment should include an analysis of severity based on symptoms and exacerbations, and on lung function (see **Table 2**).^{4,6,16}

Research Review Educational Series

Managing chronic obstructive pulmonary disease (COPD) in general practice

Table 5. Subsidised inhaled medications for the treatment of stable COPD in New Zealand^{1,16,37,38} The newly funded medicines 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 Budenocort	100-800 mcg Max dose: 800 mcg twice daily	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
	Fluticasone propionate + salmeterol	Vannair	200 + 6 mcg Two puffs Max dose: 4 puffs daily	Twice daily	MDI	Fully subsidised
		Seretide; Rexair	125 + 25 mcg Two puffs	Twice daily	MDI – spacer recommended	Fully subsidised
	Fluticasone furoate + vilanterol	Seretide Accuhaler	250 + 50 mcg One puff Max dose: 500 + 50 mcg twice daily	Twice daily	DPI breath-activated [Accuhaler]	Fully subsidised
		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.

EXPERTS' CONCLUDING REMARKS

Concluding remarks from Jim Reid:

COPD is a disease that doctors can really do something about. It is a condition that should be diagnosed early in the disease process and addressed in primary care, with smoking cessation and early referral to pulmonary rehab at the top of the "treatment" priorities. All patients should have a management plan instructing them what to do and when to do it in the case of an exacerbation. In addition, the plan should include current therapy, FEV₁ and saturations when the patient is well, as well as whether they are a CO₂ retainer. Such information is crucial to ambulance and ED staff if they are involved in an emergency exacerbation.

The addition of a number of new inhalers to the armamentarium is great news, but one thing is important – it is crucial to standardise as far as possible the type of inhaler used and thus the brand, all of which are different. To be using two or three different types of inhaler only leads to confusion.

Concluding remarks from Lutz Beekert:

It is great to have more medications available to assist our patients with COPD. Most of the new medications are further developments of current inhalers with good safety records. Most of the single and combination inhalers improve symptoms; however, disappointingly none have been shown to improve survival. New Zealand is lucky to have well-informed, compassionate General Practitioners who deliver excellent COPD care. We need to continue to improve access to pulmonary rehabilitation programmes and treat 'smoking as the disease instead of waiting for COPD'. New Zealand should become virtually Smokefree by 2025; this will eventually cure most of COPD – at the moment we have some new treatment options, let's use them responsibly.

Useful resources:

BPAC New Zealand COPD Prescribing Tool. 2016. Available from <http://www.bpac.org.nz/2016/copd-tool/>

Lung Foundation Australia. Available from: <http://lungfoundation.com.au/>

The COPD-X Plan. Australian and New Zealand Guidelines for the management of Chronic Pulmonary Disease. 2015. Available from: <http://copdx.org.au/copd-x-plan/>

Global Initiative for Chronic Obstructive Lung Disease (GOLD). Global strategy for the diagnosis, management and prevention of COPD. Updated 2016. Available from: <http://www.goldcopd.org>

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