Managing COPD in the new millennium

Chronic obstructive pulmonary disease (COPD) is the third leading cause of disease burden in Australia, but management of the condition is far from optimal. Increased awareness of the disease and adherence to published guidelines for diagnosis and treatment are needed to meet the goals of improving patients' quality of life and avoiding hospitalisation.

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Chronic obstructive pulmonary disease (COPD) is defined as airflow limitation due to narrowing of large and/or small airways without significant bronchodilator reversibility. Emphysema is characterised by enlargement of air spaces and destruction of lung parenchyma, resulting in loss of lung elasticity and contributing to closure of small airways and gas trapping. Patients with COPD also frequently have symptoms of chronic bronchitis (cough and sputum production).

According to the World Health Organization (WHO), by 2020 COPD will be the world's fifth leading cause of disease burden, as measured in disability-adjusted life years (that is, the sum of years lost by premature mortality plus years lived with disability, adjusted for the severity of the disability). It is currently the third leading cause of disease burden and fourth leading cause of death in Australia.[1] COPD is a major burden in terms of the costs involved in treatment, hospital admissions, and loss of productivity. The Australian Lung Foundation's Chronic Airflow Limitation Consultative Group recently estimated the total direct and indirect costs of COPD to the community to be approximately $800 million annually, with more than 20,000 new cases diagnosed each year.[1,2]

Risk factors

In developed countries, smoking is the major risk factor for COPD. Normal airway function is expected to decline with age at a rate of less than 30 mL per year, a rate that may increase to more

IN SUMMARY

- **A diagnosis of COPD should be considered in any patient who has a smoking history of more than 10 pack-years, especially if typical symptoms such as chronic cough, sputum production or dyspnoea are present.**
- **Spirometry is mandatory to confirm a suspected diagnosis of COPD and to exclude asthma.**
- **Smoking cessation is the single most effective measure for preventing development of COPD and slowing its progression. Patients should be informed that it is never too late to stop smoking.**
- **Although there is evidence to support the use of pulmonary rehabilitation programs for patients with COPD, such programs are highly underutilised. All regional health services should be able to organise access to a pulmonary rehabilitation program.**
- **Management of COPD is suboptimal. Poor adherence to published guidelines for diagnosis and management and patient noncompliance are contributing factors.**
- **New and exciting treatments for COPD may be not far over the horizon. These will improve our existing therapies but will need to be used judiciously.**

continued

Table. Classification of COPD severity*

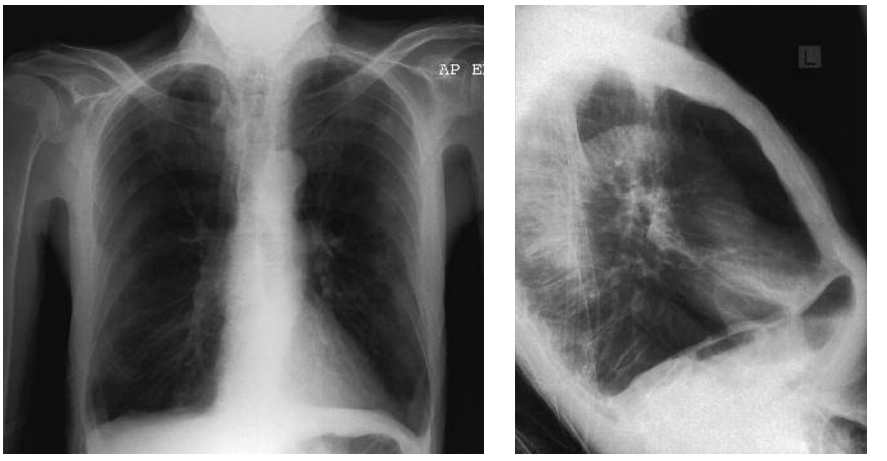
Stage 0: at risk
- Normal spirometry
- Chronic symptoms (cough, sputum)

Stage I: mild
- FEV_1/FVC <70%
- FEV_1 ≥80% predicted
- Symptoms present or absent (cough, sputum)

Stage II: moderate
- FEV_1/FVC <70%
- 30% ≤FEV_1 <80% predicted
  Stage IIA: 50% ≤FEV_1 <80% predicted
  Stage IIB: 30% ≤FEV_1 <50% predicted
- Chronic symptoms present or absent (cough, sputum, dyspnoea)

Stage III: severe
- FEV_1/FVC <70%
- Either FEV_1 <30% predicted or FEV_1 <50% predicted with respiratory failure or clinical signs of right heart failure

* Adapted from the GOLD guidelines, *Global initiative for chronic Obstructive Lung Disease* (see reference 4).



Figures 1a and b. Chest x-rays of a 75-year-old man with COPD taken in the emergency department. a (left). Anterior-posterior view showing hyperinflated lungs with flattened diaphragm. b (right). Lateral view showing increased anterior–posterior diameter (barrel chest), increased size of the retrosternal window and flattened diaphragm. There is osteopenia in the vertebral column with a crush fracture at T7 and loss of vertebral height at multiple levels secondary to frequent use of prednisolone for acute exacerbations.

than 100 mL per year in smokers. Other environmental factors may contribute to development of the disease, such as heavy exposure to occupational dusts and chemicals and air pollution.

Diagnosis

A diagnosis of COPD should be considered in any patient who has a smoking history of more than 10 pack-years (one pack-year is defined to be 20 cigarettes per day for a year or an equivalent total number of cigarettes), especially if typical symptoms are present, such as chronic cough, sputum production or dyspnoea.

It is estimated that only 15 to 20% of smokers develop disabling COPD, but the true figure may well be higher because some patients may be misdiagnosed with asthma – community studies have suggested that primary care physicians may have difficulties in distinguishing between these two conditions.[3] However, there is some overlap: some patients with asthma show chronic airflow obstruction and limited reversibility and some COPD patients show a degree of bronchodilator reversibility.

Spirometry

Spirometry is mandatory to confirm a suspected diagnosis of COPD and to exclude asthma. Patients with COPD may be asymptomatic until their FEV_1 has fallen below 50 to 60% of predicted values or below 1.5 L.[4] Patients often attribute their progressive impairment in exercise tolerance to ageing and are diagnosed with the condition relatively late. The severity of COPD is conventionally classified according to spirometric findings and the presence or absence of cough or breathlessness (Table).

The use of spirometry in diagnosis of COPD is discussed further in the box on page 32.

Imaging

Features on chest x-ray such as hyper - inflation of the lungs, flattening of the diaphragm and attenuation of vascular markings may suggest the presence of emphysema (see Figures 1a and b). High resolution CT scanning is very sensitive in the detection of emphysema (Figure 2).

Measures for disease prevention and modification

Nonpharmacological treatments such as smoking cessation and long term home oxygen have been shown to improve sur-

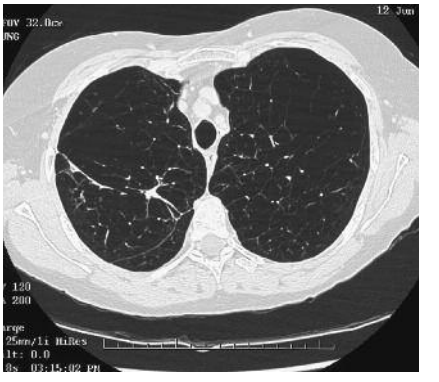


Figure 2. High resolution chest CT scan of a 70-year-old woman with emphysema. Extensive emphysematous changes with bullae are visible in both lungs.

vival in COPD patients. Pulmonary rehabilitation is effective in improving patients' exercise tolerance, breathlessness, confidence and quality of life.[5,6] On the other hand, pharmacological treatments such as short and long acting bronchodilators, methylxanthines, and inhaled and systemic corticosteroids have not been shown to be effective in reducing mortality or the rate of decline in lung function in patients with COPD.

Smoking cessation

Smoking cessation is the single most effective measure for preventing development of COPD and slowing progression. Patients should be informed that it is never too late to stop smoking and that the rate of decline of their lung function will return to that of nonsmokers when they stop.

Professional psychological support is as important as use of pharmacological interventions such as nicotine replacement therapies (NicabateCQ, Nicorette, Nicotinell, QuitX) and bupropion (Zyban SR). The combination of non-pharmacological and pharmacological treatment modalities increases the chance of successful quitting by 5 to 30%.[7]

Long term oxygen therapy

Long term use of home oxygen for more than 15 hours a day in patients with severe hypoxaemia has been shown to improve length and quality of life. It is indicated in patients with COPD who are not smoking and have arterial oxygen tension (PaO_2) of less than 55 mmHg or oxygen saturation (SaO_2) of less than 88%.[8] Long term oxygen therapy is also indicated in patients with less severe hypoxaemia (PaO_2 55 to 60 mmHg) in the presence of right heart failure, pulmonary hypertension and polycythaemia.

Pulmonary rehabilitation

Pulmonary rehabilitation focuses on improving patients' self-efficacy as well as optimising current management and physical function. There is a large body of evidence that pulmonary rehabilitation results in improved fitness, wellbeing and quality of life and decreased consumption of healthcare resources by reducing acute attendances at emergency departments and hospital admissions. In spite of this evidence, there are data showing that only 1 to 2% of patients with COPD are enrolled into pulmonary rehabilitation programs each year.[9]

Pulmonary rehabilitation programs involve multidisciplinary approaches to exercise training and patient education. A basic program will include education on medication, sputum clearance, relief of shortness of breath and community support services, as well as training in use of inhalation devices, relaxation techniques, energy conservation, long term oxygen therapy, and cardiovascular exercise training. The content of a program will depend on the resources available.

All regional health services should be able to organise access to a pulmonary rehabilitation program, either locally or through a regional reference point. GPs can contact the respiratory or physiotherapy department of their local hospital for details and referral procedures.

Guidelines for managing COPD

Guidelines for treating COPD have been produced by various thoracic societies. The 'GOLD' guidelines developed by the US National Heart, Lung, and Blood Institute and WHO, *Global initiative for chronic obstructive lung disease*,[4] addresses disease assessment and monitoring, reduction of risk factors, management of stable COPD, and management of exacerbations. GOLD documents and resources are available on the internet (www.goldcopd.com).

The Australian and New Zealand management guidelines of COPD is a joint project of the Thoracic Society of Australia and New Zealand and the Australian Lung Foundation. A draft of the guidelines is available to GPs (www.lungnet.com.au). These guidelines are presented as the COPD-X plan:

C – Confirm diagnosis and assess severity
O – Optimise function
P – Prevent deterioration
D – Develop support network and self-management plan
X – manage acute eXacerbations appropriately.

The Australian and New Zealand guidelines are expected to be released in 2003.

Management of stable disease

Management of COPD is suboptimal. Use of clinically proven effective treatments is mandatory, and should be in accordance with published guidelines. Some of these are discussed in the box on this page.

Short-acting bronchodilators

The GOLD guidelines (*Global initiative for chronic Obstructive Lung Disease*) recommend use of inhaled short-acting bronchodilators such as salbutamol, terbutaline (Bricanyl for Inhalation) and ipratropium bromide in COPD patients, even if spirometry shows no significant airway reversibility following use of aerosol bronchodilators. These agents provide symptomatic relief and improve quality of life. Combined use of salbutamol and ipratropium bromide is more effective than either agent alone.

Inhaled corticosteroids

Inhaled corticosteroids should be reserved for patients with objective improvement of airway function (corticosteroid reversibility) after a trial of six weeks to three months. Corticosteroid reversibility is defined as an increase in FEV_1 of 200 mL

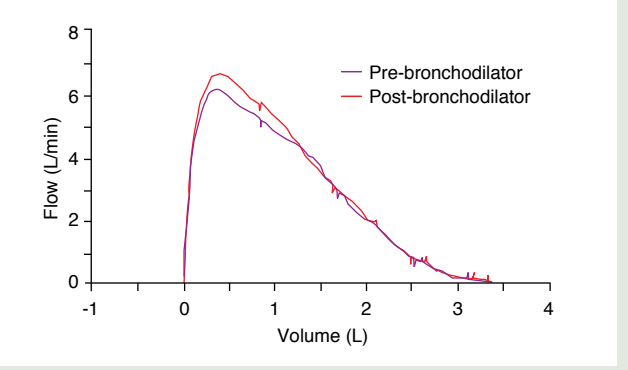
Using spirometry to diagnose COPD

Spirometry is mandatory to confirm a diagnosis of COPD. The typical spirometric findings are:

- a FEV_1 of less than 80% of the predicted value, and
- a reduction in the ratio of FEV_1/FVC to less than 70% post-bronchodilator.*

The presence of significant reversibility with a bronchodilator is suggestive of asthma. Significant reversibility is defined as an improvement in FEV_1 of more than 15% if the FEV_1 is greater than 1.5 L or more than 200 mL if the FEV_1 is less than 1.5 L.

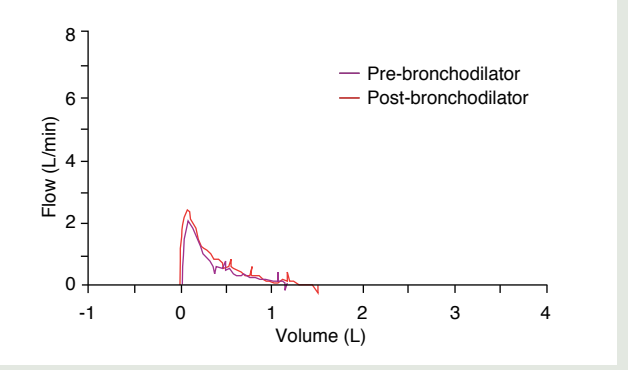
Normal spirometry



	FEV_1	FVC	FEV_1/FVC
Pre-bronchodilator			
Measured value	2.63 L	3.40 L	77%
Measured value ÷ predicted value	105%	116%	–
Post-bronchodilator			
Measured value	2.66 L	3.33 L	80%
Measured value ÷ predicted value	106%	114%	–
Change with bronchodilator			
Absolute change	30 mL	-70 mL	–
% change (absolute change ÷ pre-bronchodilator measured value	1%	-2%	–

Figure A. Normal expiratory flow–volume curve.

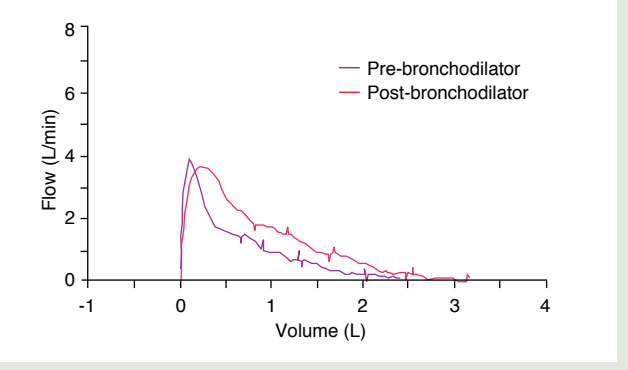
Irreversible airway obstruction



	FEV_1	FVC	FEV_1/FVC
Pre-bronchodilator			
Measured value	0.74 L	1.56 L	47%
Measured value ÷ predicted value	45%	78%	–
Post-bronchodilator			
Measured value	0.78 L	1.92 L	41%
Measured value ÷ predicted value	48%	96%	–
Change with bronchodilator			
Absolute change	40 mL	360 mL	–
% change (absolute change ÷ pre-bronchodilator measured value	6%	23%	–

Figure B. Expiratory flow–volume curve showing irreversible airway obstruction. The reduced pre-bronchodilator FEV_1 (<80% predicted) and insignificant FEV_1 reversibility (<200 mL) are consistent with a diagnosis of COPD.

Reversible airway obstruction



	FEV_1	FVC	FEV_1/FVC
Pre-bronchodilator			
Measured value	1.31 L	2.70 L	48%
Measured value ÷ predicted value	45%	75%	–
Post-bronchodilator			
Measured value	1.71 L	3.30 L	52%
Measured value ÷ predicted value	63%	92%	–
Change with bronchodilator			
Absolute change	400 mL	600 mL	–
% change (absolute change ÷ pre-bronchodilator measured value	31%	22%	–

Figure C. Expiratory flow–volume curve showing reversible airway obstruction. The reduced pre-bronchodilator FEV_1 (<80% predicted), with significant bronchodilator reversibility (>200 mL) is suggestive of asthma.

* FEV_1 = forced expiratory volume in one second; FVC = forced vital capacity. Predicted values are based on age, gender and height.

and 15% above baseline.[4] Recent studies have suggested that inhaled corticosteroids may also be indicated in patients who have moderate to severe COPD (FEV_1 less than 50% predicted), especially those who have frequent acute exacerbations requiring antibiotics and systemic corticosteroids.[4,10] The use of inhaled corticosteroids in this group of COPD patients may reduce the number of acute exacerbations but has no effect on lung function or survival.

Mucolytics

Mucolytics such as bromhexine (Bisolvon Chesty Oral Liquid, Bisolvon Chesty Tablets, Duro-Tuss Mucolytic Cough Liquid) and acetylcysteine (Mucomyst) may play a role in COPD patients who have difficulty expectorating tenacious sputum. A Cochrane review concluded that mucolytic agents provide a small reduction in acute exacerbations and the total number of days of disability.[11]

Other measures

Other measures that should not be overlooked for COPD include vaccinating against influenza, treating depression and correcting malnutrition. Low body mass index (BMI) and weight loss are two important adverse prognostic factors for patients with COPD. The role of lung volume reduction surgery in managing COPD is currently being examined in a large clinical trial.[12]

Management of acute exacerbations

Acute exacerbations of COPD refer to worsening of previously stable situations. Patients usually present with increasing shortness of breath, cough with sputum, wheeze, hypoxia, and increased airway obstruction as measured by spirometry. The economical and social burden of these exacerbations is extremely high.

The most common causes of acute exacerbations of COPD are respiratory infections (both upper and lower respiratory tracts) and air pollution,[13] but no cause is found in 30% of cases. Other conditions that may mimic acute exacerbations must be excluded, including pneumothorax, pulmonary embolism, pleural effusion, cardiac failure and arrhythmia.

Treatments for acute exacerbations, which are further discussed below, consist of systemic corticosteroids, appropriate antibiotics and assisted ventilation in hypercapnic respiratory failure. Bronchodilators and controlled oxygen therapy (with target SaO_2 of 90%) also play a role.

Systemic corticosteroids

Oral and intravenous corticosteroids are recommended in acute exacerbations of COPD. A course of oral corticosteroids administered during the first 72 hours speeds recovery and reduces the duration of hospitalisation.[14] Between 30 and 40 mg of oral prednisolone (Panafcortelone, Solone) daily for 10 to 14 days is a reasonable dose. Prolonging the course of treatment beyond two weeks does not result in further benefit, and the risk of side effects such as hyperglycaemia increases.

Antibiotics

Antibiotics are only effective when a patient presents with increasing cough and dyspnoea as well as increased sputum volume and purulence.[15] Fever and leucocytosis are also suggestive of an underlying infective bacterial cause for the exacerbation. The major organisms associated with acute exacerbations are *Haemophilus influenzae*, *Streptococcus pneumoniae* and *Moraxella catarrhalis*. *Mycoplasma pneumoniae* and *Chlamydia pneumoniae* may also contribute.

Amoxycillin, doxycycline or a macrolide (erythromycin or roxithromycin [Biaxsig, Rulide]) can be used initially, but amoxycillin–clavulanate should be prescribed if patients do not respond. Typically, a course of 10 days is needed. Intravenous antibiotics are indicated in those patients who are septic, unable to tolerate oral intake or critically ill.

Assisted ventilation

Noninvasive positive pressure ventilation is an effective and safe means of treatment in patients with COPD who have acute hypercapnic respiratory failure. Randomised controlled trials have shown that fewer hypercapnic respiratory failure patients require intubation, and complication and mortality rates also decrease. The success rate is as high as 80%.[16] The decision to intubate in patients with respiratory failure who fail noninvasive positive pressure ventilation is influenced by the likely reversibility of the precipitating event and the patient's wishes.

Barriers to successful management

There are currently two important barriers to successful management of COPD:

- poor physician adherence to treatment guidelines
- patient noncompliance.

A study of French respiratory physicians' adherence to COPD guidelines versus clinical practice published in 2001 found that 78% of COPD patients were treated with β_2-agonists, 56% with anticholinergic agents, 31% with methyl xanthines, and 76% with inhaled corticosteroids.[17] The high proportion of prescriptions for inhaled corticosteroids and the predominance of β_2-agonists over anticholinergic agents contrast with the current European recommendations for management. Overprescribing inhaled corticosteroids may be related to misdiagnosing COPD as asthma, and could be a significant source of unnecessary expense and adverse effects. It has been estimated that misuse of inhaled corticosteroids may cost the British National Health Service £42 million per annum.[3] There are no direct data in Australia, but clinical impressions are similar.

Several studies have found patient

compliance with COPD therapies to be poor. For example, self-reported patient compliance with inhaled bronchodilator therapy in the Lung Health Study was just over 60% at one year, declining to less than 50% at the end of five years.[18] Non-compliance may increase the frequency of primary care visits and possibly hospital admissions. Therefore, new effective treatments are needed that pay attention to patient compliance.

Newer developments in pharmacotherapy

Recently, much more research effort has been put into modifying the underlying airway inflammation in COPD, which is very different to that of asthma.[19] (Eosinophils, mast cells and CD4+ lymphocytes are the principal inflammatory cells in asthma, whereas neutrophils and macrophages play a major role in COPD.) There are promising new groups that have evidence of clinical utility to support their use in COPD, although they are either not yet available or not registered for this use in Australia.

Long-acting anticholinergic agents

Anticholinergic agents (e.g. ipratropium, a short-acting agent) have been shown to be effective for improving dyspnoea and exercise tolerance in patients with COPD. Vagal cholinergic tone appears to be an important potentially reversible component of airway narrowing in this condition. There are three subtypes of muscarinic receptors in human airways:

- M1 receptors, which are located at the parasympathetic ganglia and facilitate cholinergic neurotransmission
- M2 receptors, which are located at cholinergic nerve endings and inhibit release of acetylcholine (i.e. act as negative feedback receptors)
- M3 receptors, which are located at airway smooth muscle cells and submucosal gland cells and mediate bronchospasm and hypersecretion.

A new long-acting anticholinergic agent which is expected to be available on the PBS in 2003, tiotropium, has great affinity for the muscarinic receptors (M1, M2 and M3). Its binding affinity is similar for the three types of receptors, but it dissociates very slowly from M1 and M3 receptors (which results in prolonged bronchodilation) and rapidly from M2 receptors (therefore reducing acetylcholine release and hence less bronchoconstriction). A 12-month, randomised, placebo-controlled trial has shown that tiotropium significantly improved the trough and average FEV_1 by 12% (120 mL) and 22% (240 mL), respectively, in patients who had moderate to severe

COPD; the FVC response parallelled those of FEV_1.[20] Patients who were in the tiotropium group felt significantly less dyspnoeic and wheezy, and their 'as needed' use of short-acting β_2-agonists was reduced. Tiotropium was also associated with fewer exacerbations and hospitalisations, as well as prolonging time to the first exacerbation, and patients' overall health status was improved relative to the placebo group. The most common side effect of treatment was a dry mouth (16% of patients).

In another randomised and placebo-controlled trial, tiotropium given once daily was shown to be more effective in improving airway function and vital capacity than ipratropium bromide given four times daily.[21] The incidence of dry mouth was similar in both treatments.

The dose of tiotropium used in both of the studies was 18 μg daily; higher doses have not been shown to produce greater spirometric improvement.[22] Significant bronchodilation is produced within an hour after the first dose, and peak spirometric improvements in FEV_1 and FVC are reached after one week of therapy. The half-life of tiotropium is about nine hours (ipratropium has a half-life of 1.5 hours), which allows once daily use,[23] and may improve patient compliance. Tiotropium as maintenance therapy for patients with moderate to severe COPD should offer significant advantages over existing bronchodilation therapy.

Long-acting β_2-agonists

Although the short-acting β_2-agonists undoubtedly play an indispensable role in reversible airway obstruction, their use in COPD is, as outlined by various guidelines, predominantly for improvement of dyspnoea. Long-acting β_2-agonists may have a different role in COPD.

There are two long-acting β_2-agonists available for use in COPD: eformoterol (Foradile) and salmeterol (Serevent). The pharmacological and clinical profiles of these two agents are slightly different. Eformoterol is a full β_2-agonist with a rapid onset of action (within minutes) comparable to that of salbutamol; salmeterol is a partial β_2-agonist (in relation to eformoterol), which explains its lesser degree of maximal bronchodilation.

The efficacy of salmeterol in COPD of moderate severity has been assessed in two trials.[24,25] Salmeterol provided significant improvements in FEV_1 (200 mL) and FVC (300 mL) from baseline, with no tolerance of its bronchodilatory effect demonstrated. A delay in the onset of exacerbations, reduced use of salbutamol, dyspnoea and improved quality of

life were also observed.[26] The efficacy of salmeterol was comparable to that of ipratropium in both studies, but salmeterol's twice daily dosing may improve patient compliance.

Long-acting β_2-agonists and corticosteroids in combination

Combining a long-acting β_2-agonist with an inhaled corticosteroid has gained wide acceptance in the management of asthma. Does inhaled β_2-agonist–corticosteroid combination therapy have a role in the treatment of COPD?

The role of inhaled corticosteroids for COPD has not been well defined. Four large, placebo-controlled studies[10,27-29] have addressed the efficacy of inhaled corticosteroids in retarding the rate of decline of FEV_1 in COPD patients over two to three years, and none of these was able to show any favourable effect on decline of airway function. However, the ISOLDE study showed that use of inhaled fluticasone propionate (which is marketed in Australia as Flixotide), 500 μg twice daily for three years, resulted in fewer exacerbations, reduced the rate of decline in health status, and produced a small initial increase in FEV_1.[10]

Results of a randomised trial showed that a combination of fluticasone (500 μg) and salmeterol (50 μg) provided significant improvements in pre-dose FEV_1 and dyspnoea compared with either fluticasone (500 μg) or salmeterol (50 μg) alone, or with placebo.[30] It also significantly improved the two-hour post-dose FEV_1 value, and reduced the need for supplemental salbutamol compared with fluticasone and placebo. A combined preparation of fluticasone and salmeterol is available (Seretide), but it is not yet approved in Australia for COPD.

Similarly, results of a double-blind placebo-controlled study (published in abstract form) have shown that the combination of budesonide and eformoterol is able to improve health status and symptoms and reduce mild and severe exacerbations in patients with moderate to severe COPD.[31,32] A combined preparation of budesonide and eformoterol is not yet available in Australia.

Phosphodiesterase 4 inhibitors

Theophylline (Nuelin), which has been used as a bronchodilator for COPD and asthma for more than 80 years, has a moderate ability to bronchodilate (improving FEV_1 of about 10%). Studies of COPD patients have shown that theophylline improves exercise tolerance, dyspnoea and wellbeing, possibly by improving peripheral ventilation and gas exchange and reducing the residual volume (allowing the diaphragm to resume its dome shape and increase its contractility).[33,34] The way that theophylline works is poorly defined, but several mechanisms have been suggested, including phosphodiesterase inhibition.

Phosphodiesterase 4 is the predominant phosphodiesterase in inflammatory cells and smooth muscle cells in the airways; inhibition of this enzyme suppresses the inflammatory cells and relaxes airway smooth muscles. Phosphodiesterase 4 inhibitors have also been shown to reduce the protease burden associated with neutrophilic inflammation, as well as down-regulating the activity of CD8+ cells and macrophages, the predominant cells causing airway inflammation in COPD.[35] Such effects have the potential to slow the accelerated decline in lung function.

First generation phosphodiesterase 4 inhibitors, such as rolipram and denbufylline, have impressive activity across a wide range of animal models of pulmonary inflammation.[36,37] However, the high incidence of side effects (nausea, vomiting and dyspepsia) render them clinically impractical for treating airway inflammation.

Second generation phosphodiesterase 4 inhibitors have markedly decreased potency for the form of phosphodiesterase 4 that predominates in the central nervous system and parietal glands but remain active against the form that predominates in inflammatory cells. They do not cross the blood–brain barrier and they have far fewer side effects than the first generation compounds. Three second generation phosphodiesterase 4 inhibitors have been developed for COPD: cilomilast, roflumilast and BAY19-8004.

The efficacy of cilomilast for moderate to severe COPD has been evaluated.[38] At an oral dose of 15 mg twice daily, trough FEV_1 was observed to rise within a week of treatment and reach a maximum on week six, averaging 160 mL more than the placebo group. Improvements were also observed in trough FVC, peak expiratory flow rate, exertional dyspnoea, rescue bronchodilator use, and the resting and post-exercise oxygen saturation. No serious adverse effects were observed in a group of healthy volunteers given cilomilast at a dose ranging from 2 to 20 mg twice daily for six days.[39] The efficacy of roflumilast has been reported in a phase II study of asthma patients. However, clinical trial results of roflumilast and BAY19-8004 for treating COPD have not yet been published.[35]

Consultant's comment

COPD is common, and it is a huge personal and cost burden in Australia. Smoking control is the major primary and secondary prevention strategy.

The Australian Lung Foundation, with The Thoracic Society of Australia and New Zealand, has used the global guidelines (GOLD) with updated evidence to formulate a set of tools to help GPs manage the condition with more confidence. Current handbooks and guides are available on the internet (www.lungnet.com.au/copd.html), and specific GP guides (known as COPD-X) will progressively be released in early 2003.

Emphasis must be given to early diagnosis. Doctors should remain alert to undue breathlessness with activities or daily cough, especially in people who have a smoking history. The presence of airflow limitation can only be demonstrated with spirometry. Early smoking cessation and encouragement of physical activity are likely to reduce or at least slow the onset of disability and burden.

At present, drug therapies cannot cure COPD but they do provide some symptom relief, and promising new drugs may control symptoms for longer periods. Pulmonary rehabilitation helps people with established COPD to live more comfortably with the condition, which is the desired goal of management. This article provides useful insights into current and accumulating evidence about COPD and its diagnosis and treatment.

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Conclusion

Management of COPD is suboptimal. The lack of awareness of COPD and its burden (by both the public and medical practitioners), as well as poor adherence to published guidelines for diagnosis and management by physicians and patient noncompliance contribute to suboptimal management. Proper diagnosis and practising of evidence-based medicine together with prescribing clinically proven effective treatments are mandatory to improve patient outcomes and make better use of government funding.

The goals of improving quality of life and avoiding hospitalisation for COPD patients are best achieved by taking a multidisciplinary approach that involves physiotherapists, occupational therapists, clinical psychologists and community nurses. GPs play a pivotal role in managing COPD patients in the community – particularly in regard to early diagnosis and the initiation of smoking cessation. Mild exacerbations can be managed at home under the supervision of a GP; hospital admissions should be reserved for severely ill patients requiring supplemental oxygen, intravenous antibiotics, systemic corticosteroids and assisted ventilation.

New and exciting treatments for COPD may be not far over the horizon. These will improve our existing therapies, but will need to be used judiciously. MT

A list of references is available on request to the editorial office.

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Since the time of writing this article, Dr Lim has taken up a senior appointment with GlaxoSmithKline in the United Kingdom.

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