

COPD

POEMs - Patient Oriented
Evidence that Matters

COPD POEMs

Patient Oriented Evidence that Matters

The aim of these POEMs is to increase the safety and effectiveness of treatment for people with COPD by promoting primary care interventions which are evidence-based, patient-centred, cost-effective and context-sensitive.

Compiled by: Dr Trevor Walker
David Woods

Reviewed by: Professor Ian Town
Dr Neil Whittaker
Dr Peter Martin

bpac^{nz} Clinical Advisory Group:
Judi Dennis
Rachael MacKay
Dr Peter Moodie
Associate Professor Jim Reid
Dr David Reith
Professor Murray Tilyard

Developed by bpac^{nz}
Level 8, 10 George Street
PO Box 6032
Dunedin
Phone 03 477 5418
Fax 03 477 2622
www.bpac.org.nz

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All information is intended for use by competent health care professionals and should be utilised in conjunction with pertinent clinical data.

Introduction

The burden of COPD

COPD imposes a significant personal, societal and financial burden on New Zealanders. In 1997, COPD was ranked third overall in its impact on the health of New Zealanders after ischaemic heart disease and stroke. It ranked second in men and fifth in women and was the fourth most common cause of death after cancer, heart disease and stroke.

In New Zealand it is probable that:

- Approximately 50,000 people aged >40 years have diagnosed COPD, and
- Approximately 180,000 people aged >40 years have undiagnosed COPD (Broad, 2003).

Tobacco smoking causes almost all COPD

Tobacco smoking causes approximately 85% of all cases of COPD. Half of all smokers are susceptible to the effects of tobacco smoke and will develop some limitation of airflow during their lifetime.

Occupational factors

There are significant associations between occupations and the risk of respiratory symptoms and physiological abnormalities suggestive of early COPD. These occupations include food processors, bakers, chemical processors, spray painters and those who report occupational exposure to vapours, gases, dust or fumes.

It is likely that these occupational environments are risk factors for COPD, although the relative importance of the occupation weighed against other risk factors (such as pollution and cigarette smoke) will vary from worker to worker and industry to industry (Fishwick, 1997).

Other factors

A small percentage of COPD sufferers will have a genetic predisposition, such as alpha-1 antitrypsin deficiency, and this should be suspected if a young person develops COPD or someone develops COPD in the absence of a significant smoking history.

In developing countries a frequent cause of COPD is indoor pollution. Often women are affected as they spend time cooking over open fires in confined spaces.

Summary

Treatment for those with COPD can be improved by adoption of the **COPDX** plan (see page 3). The following actions are critical to the success of the plan:

1. Earlier diagnosis for those with COPD

Steps to earlier diagnosis include identifying all smokers enrolled with the practice, screening them ideally with spirometry or possibly PEFR and using spirometry to ensure an accurate diagnosis.

2. Increased use of the combined approach of nicotine replacement therapy (NRT) and smoking cessation programmes for smokers

Smoking cessation is the only intervention shown to halt the accelerated decline in lung function seen in COPD. This combined approach to smoking cessation has proven effectiveness.

3. Limitation of the inappropriate use of steroids (inhaled and oral) by those with COPD

Oral steroids are useful only in the management of COPD exacerbations. Inhaled steroids have a very limited role and are not approved for use in COPD in many countries.

4. Use of a stepped regime of bronchodilator therapy for those with COPD

A suitable stepped regime is one such as:

1. Intermittent inhaled short acting beta-2 agonist.
2. Addition of regular combined short acting beta-2 agonist plus ipratropium.
3. Tiotropium for severe COPD.

5. Appropriate use of antibiotics in the treatment of COPD

Antibiotics are only useful in exacerbations with evidence of bacterial infection. Prophylactic therapy is not helpful.

6. Increased uptake of annual influenza and five-yearly pneumococcal vaccination by those with COPD

The strongest evidence is for influenza immunisation, although pneumococcal immunisation is likely to be beneficial.

7. Increased use of pulmonary rehabilitation in the treatment of those with moderate to severe COPD

Pulmonary rehabilitation should be offered to all those with moderate or severe COPD.

The COPDX Plan

The COPDX plan has been developed by The Australian Lung Foundation and The Thoracic Society of Australia and New Zealand, and can be accessed in full on the New Zealand Guidelines Group website (NZGG, 2002). A summary of the COPDX plan has been produced and circulated to practitioners in 2004.

The key components of the COPDX plan are summarised as follows:

C	Confirm diagnosis & assess severity by use of spirometry and measurements of functional impairment.
O	Optimise function by relief of symptoms, increasing wellbeing and reducing the number and severity of exacerbations and complications.
P	Prevent deterioration by smoking cessation and reduction of exposure to other harmful inhaled fumes and particles.
D	Develop support network and self-management plan.
X	eXacerbations - manage appropriately and promptly.

C Confirm diagnosis & assess severity

Earlier diagnosis of COPD

The key components of earlier diagnosis for COPD are:

- Identifying all smokers enrolled in the practice,
- Screening some/all smokers for airflow obstruction ideally with spirometry or possibly PEFR,
- Using spirometry to ensure an accurate diagnosis.

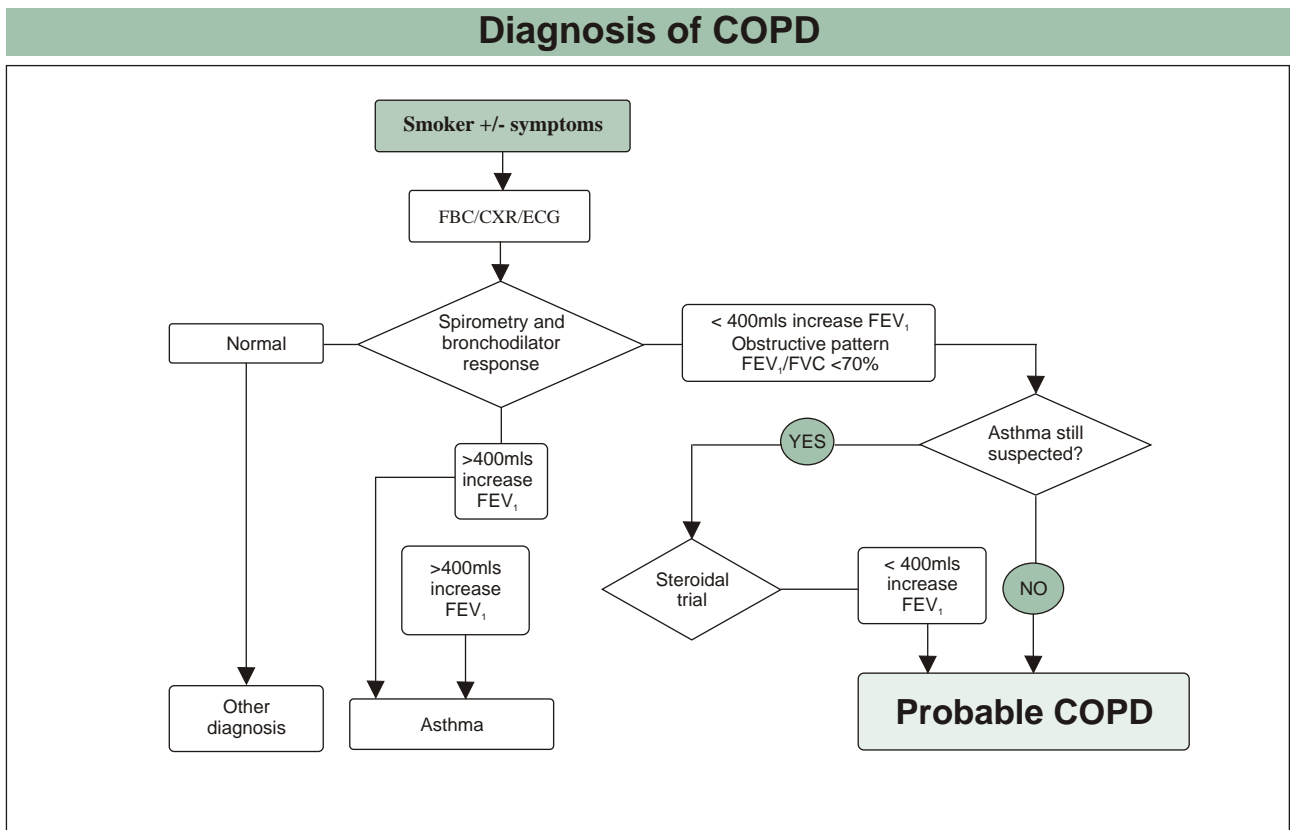
The greatest gain will be in younger patients with early clinical or spirometric features of COPD if they can be persuaded to give up smoking.

The main symptoms of COPD are breathlessness, cough and sputum production. The diagnosis should be considered in patients aged over 35 years with recurrent episodes of these symptoms, especially if they have significant exposure to tobacco smoke, pollution, occupational dusts or fumes or they have a strong family history of COPD.

Examination findings such as wheeze, hyperinflation and prolonged expiratory phase are common in COPD, however their absence does not exclude the diagnosis.

Spirometry is the gold standard for the diagnosis of COPD

The gold standard for the diagnosis, assessment and monitoring of COPD is spirometry. The use of spirometry in the diagnosis of COPD is summarised in the flow chart below.



From: The Asthma and Respiratory Foundation of New Zealand. Diagnosis & Treatment of COPD.

The role of Peak Expiratory Flow Rate (PEFR) in COPD

Although spirometry is essential in COPD there is still a role for PEFR.

There is poor correlation between PEFR and FEV₁ caused by variations in the degree of airway collapsibility between COPD patients. In addition the small changes in airway function typical of COPD are not reliably detected by PEFR.

However, a recent investigation demonstrated that a PEFR of <80% of predicted normal detected approximately 90% of patients aged between 50 and 90 years-old with COPD. The patients that were missed all had mild COPD. Of the people who had PEFR <80% of predicted normal, 17% did not have COPD (Jackson, 2003).

It seems then that PEFR may be a useful case finding tool with the proviso that we will miss some people with COPD (this study suggests they will tend to have mild COPD), and quite a few people with PEFR <80% will turn out not to have COPD. If PEFR is used for screening, those with positive or equivocal results should be referred for formal spirometry and smokers with negative results cannot be reassured that their lung function is normal.

PEFR also plays an important role when people have a mixed picture of COPD and asthma with significant reversibility of airway obstruction.

O Optimise function

Initial treatment depends on severity of COPD

FEV₁ is the strongest indicator of prognosis and best guide to initial treatment in COPD. However, functional impairment and spirometry results are not always clearly related and both must be taken into account.

COPD severity guide		
Severity	FEV ₁ *	Symptoms
At risk	Normal	Age >35yrs with past or current history of >10 pack years** smoking. Occupational exposure. Strong family history.
Mild	60% - 80%	No symptoms or breathless on strenuous exercise only (MRC1) ***. Dyspnoea when hurrying on the level or walking up a slight hill (MRC 2).
Moderate	40% - 60%	Has to walk slower than most people of the same age on the level because of breathlessness or has to stop for breath when walking at own pace on the level (MRC 3). Stops for breath after walking about 100 yards or after a few minutes on the level (MRC 4).
Severe	30% - 40%	Too breathless to leave the house or breathless when dressing (MRC 5).
Very severe	<30%	Permanent respiratory distress.

* percentage of predicted value.

** one pack year = 20 cigarettes per day for one year.

*** refers to grading of dyspnoea on the Medical Research Council (MRC) dyspnoea scale.

MRC Dyspnoea Scale	
Grade One	I only get breathless with strenuous exercise.
Grade Two	I get short of breath when hurrying on the level or up a slight hill.
Grade Three	I walk slower than people of the same age on the level because of breathlessness or have to stop for breath when walking at my own pace on the level.
Grade Four	I stop for breath after walking 100 yards or after a few minutes on the level.
Grade Five	I am too breathless to leave the house or I am breathless on dressing.

The MRC scale is a useful validated score of dyspnoea in COPD (Bestall, 1999).

The following guide to initial treatment is suggested. At follow up, treatment will need to be tailored to the needs and responses of individual patients.

Guide to initial treatment				
At risk	Mild	Moderate	Severe	Very Severe
				Oxygen and/or surgery ^{##} may be indicated.
			Tiotropium - special authority. Trial of inhaled corticosteroids. Possibly theophylline.	
		Regular inhaled short acting bronchodilator (ipratropium or beta agonist or combination of both) if responsive. Pulmonary rehabilitation. LABA may be beneficial but not currently funded for this indication.		
	Intermittent inhaled short acting bronchodilator (ipratropium or beta agonist) before exercise.			
Smoking cessation [#] - counselling plus NRT. Annual spirometry & questions regarding cough, sputum, dyspnoea.				

[#] Smoking cessation is the only intervention which slows disease progression. Other interventions are aimed at optimising function or decreasing number of exacerbations.

^{##} Surgical options may include bullectomy and lung volume reduction. Lung transplantation is usually reserved for younger patients e.g. those with alpha-1 antitrypsin deficiency.

Follow up treatment tailored to response of individual patients

In order to assess the response to treatment and enable appropriate modification we need objective measurement of change. However, treatment often does not produce measurable changes in spirometry so objective measurements of symptoms and well-being also need to be recorded.

Objective measures include:

- FEV₁ or PEFr
- Dyspnoea on the MRC scale
- Frequency and severity of cough and sputum
- Exercise capacity such as distance able to walk
- Number of exacerbations
- Smoking status

Other important areas to consider include:

- Ability to sleep, fatigue
- Psychosocial functioning
- Adherence to medication and use of inhalers
- Vaccinations due - influenza, pneumococcus
- Participation in pulmonary rehabilitation
- Self-management plan up to date
- Evidence of complications - sleep apnoea, osteoporosis, gastro-oesophageal reflux, aspiration
- Evidence of comorbidities such as heart failure

Review of medication

The response to medication is slow in COPD so a trial of at least four weeks is needed to assess response to treatment changes.

Evidence about the effectiveness of medications for COPD is often equivocal and international drug funding authorities interpret this evidence in different ways. For example inhaled corticosteroids are unlicensed for use in COPD in the UK except when combined with Long Acting Beta Agonists (LABAs), and LABAs are not funded for use in COPD in New Zealand.

bpac^{nz} suggest the following pragmatic hierarchical approach to medication use in COPD in New Zealand, however there are other valid approaches which you might decide to use for individual patients after reading the review of medication that appears later in this POEM.

A hierarchical approach to medication use in COPD	
Intermittent symptoms	Short acting inhaled beta-2 agonist as needed (e.g. salbutamol 200mcg PRN).
Persistent symptoms	Regular short acting inhaled beta-2 agonist and ipratropium (e.g. salbutamol 200-400mcg plus ipratropium 40mcg QID).
Severe COPD	Regular tiotropium 18mcg daily with inhaled beta-2 agonist as needed.
Frequent exacerbations	Trial of inhaled corticosteroids (e.g. Fluticasone 250-500mcg BD). Availability of prednisone and antibiotic at home.

The reduction in exacerbation rates achieved by inhaled corticosteroids is very modest and the results come from one study only. They should be discontinued in individual patients who do not show benefit. Because of the equivocal evidence of efficacy and the potential for side effects, inhaled corticosteroids are not approved for COPD in many countries (Cooper, 2005). Side effects include easy bruising, cataract formation and possible contribution to osteoporosis.

People with COPD are at high risk of osteoporosis because of smoking, vitamin D deficiency, low BMI, hypogonadism, sedentary lifestyle and the use of oral steroids. Although oral steroids are a known risk factor the situation with ICS is less clear. A higher risk does seem likely at high doses.

What should I do about people with COPD who have been on inhaled steroids for a long time without a documented response?

A dilemma occurs with people have been on inhaled corticosteroids (ICS) for a prolonged period of time when a search of the notes does not reveal a documented response to them. In most people the dose can be reduced and if the patient remains stable they can be discontinued. However, there is some evidence that withdrawing the steroids can result in increased breathlessness and reduced exercise tolerance (O'Brien, 2001).

We suggest that the need for ICS is reviewed for patients with COPD when presenting for repeat medications, paying particular attention to those on high daily doses of ICS (Fluticasone >500mcg daily, Beclomethasone >800mcg daily) because of their higher risk of side effects. Medications mentioned for the first time in this table are reviewed later on in this POEM.

Suggested schema for review of people with COPD on ICS
Is there documented evidence of improved:
<ul style="list-style-type: none">• FEV₁ or PEFR• Dyspnoea on the MRC scale• Frequency and severity of cough and sputum• Exercise capacity such as distance able to walk• Number of exacerbations
<ul style="list-style-type: none">• Is patient using a spacer with good technique?
<ul style="list-style-type: none">• Is a comorbidity contributing to the problem?
<ul style="list-style-type: none">• Check patients ideas and expectations of the ICS
Check that other therapy is optimum
<ul style="list-style-type: none">• Inhaled bronchodilators (short acting beta agonists, ipratropium, tiotropium)• Possibly theophylline
<ul style="list-style-type: none">• Is patient undergoing pulmonary rehabilitation?
<ul style="list-style-type: none">• Would the patient benefit from surgery or oxygen?
<ul style="list-style-type: none">• If you are satisfied with above try reducing ICS at a rate of one puff per month with careful monitoring of patient well-being.

Inhaler technique using spacer

All patients with COPD who are using metered dose inhalers should be using them through a spacer.

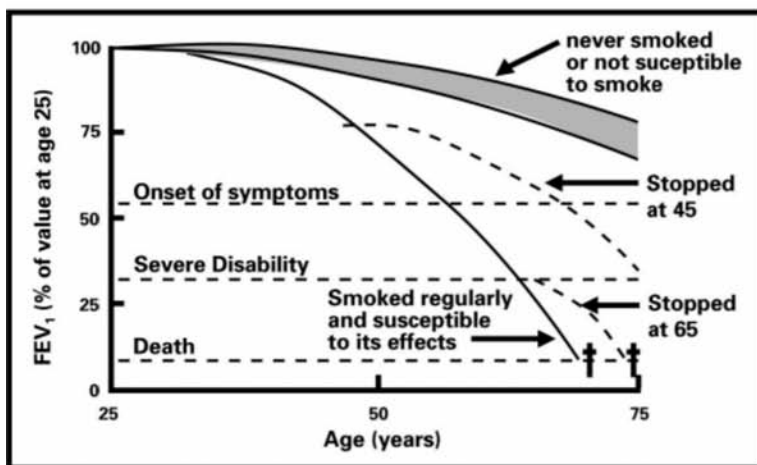
A dry powder inhaler may be a good alternative but turbuhalers should be avoided in those few patients who cannot produce inspiratory flow greater than 30L per minute as they may not achieve adequate drug delivery. This does not apply to tiotropium which is delivered through a handihaler.

P Prevent deterioration

Smoking status

Smoking cessation is the only intervention which will halt the accelerated decline in lung function seen in COPD (see diagram). It is effective at all stages of the disease.

No medication has been shown to prevent the long-term decline in lung function.

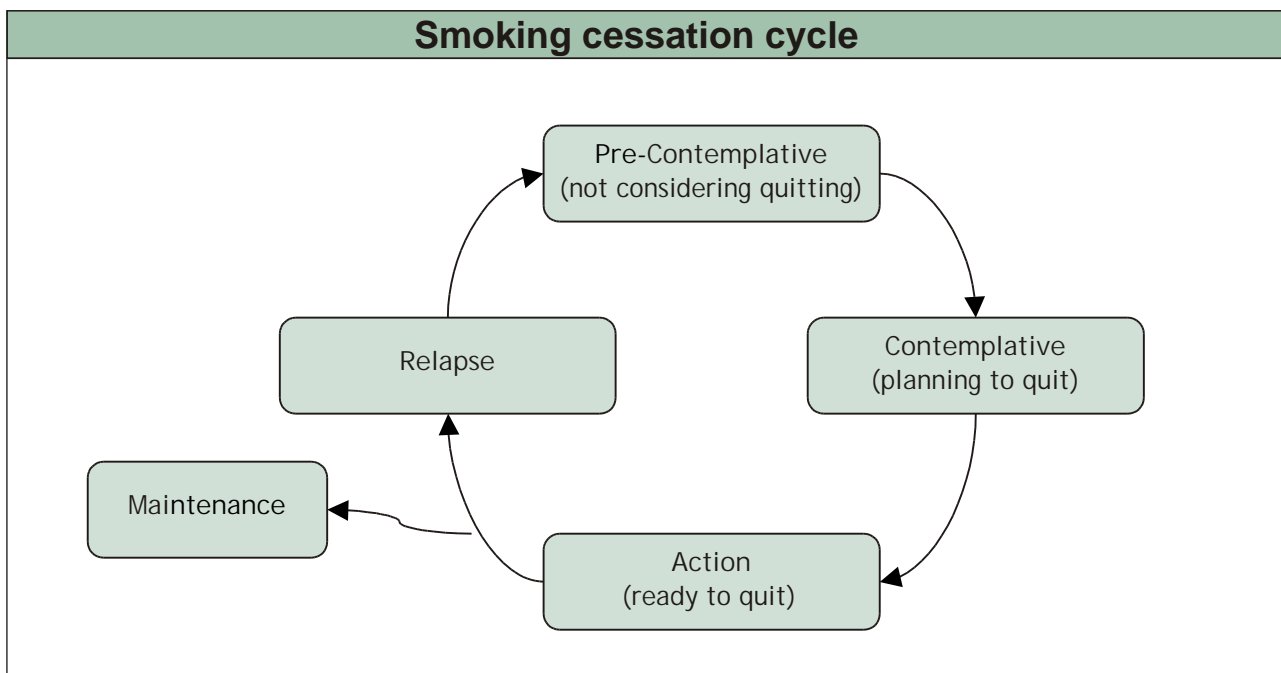


Time Course of COPD (NZGG,2002)

The risks of developing chronic obstructive lung disease: the lines illustrate the natural decline and the effects that smoking and stopping smoking can have on the FEV₁. † = death. This graph shows the rate of loss of FEV₁ for a hypothetical, susceptible smoker, and the potential effect of stopping smoking early or late in the course of COPD. Other susceptible smokers will have different rates of loss, thus reaching 'disability' at different ages. The normal FEV₁ ranges from below 80% to above 120% so this will affect the starting point for the individual's data (not shown).

Adapted from Fletcher, C & Peto, R'

The task of the clinician is to determine the patient's position on the smoking cessation cycle and use this knowledge to move the patient at least one step around the cycle.



Brief counselling is an effective way of progressing people around the cycle and should be provided at every visit. The 5-A strategy is currently accepted best practice:

- **A**sk and identify smokers.
- **A**dvice smokers about the risks of smoking and benefits of quitting and discuss options.
- **A**ssess their readiness to quit, degree of nicotine dependence and explore what would motivate them to quit.
- **A**ssist cessation this may include specific advice about pharmacological interventions or referral to a formal cessation programme.
- **A**rrange follow-up (by you or someone else) to reinforce the message.

Smoking cessation counselling is effective especially if combined with nicotine replacement therapy (NRT). This is covered in the guide for Primary Care Nurses which has been produced as a supplement to this POEM.

Recall list for influenza and pneumococcal vaccination

Annual influenza vaccination is recommended for all patients with COPD. The evidence for pneumococcal vaccination is less convincing but vaccination every five years will help prevent pneumonia in these patients with already reduced respiratory reserve.

D Develop support network & self management plan

Self-management plan

Patients with chronic illness who participate in self-management have better outcomes than those who do not. In asthma a self-management plan focuses on fine-tuning pharmacological interventions in response to day-to-day variations in lung function, however in COPD, pharmacological treatment is less effective and self-management plans require a more patient-centred approach.

A COPD self-management plan should include the following components:

- Reminder of day to day medications.
- Lifestyle tips to improve functional status and avoid exacerbations.
- Early recognition of exacerbations.
- Prompt response to exacerbations, including self-medication with prednisone and/or antibiotics.

See 'Appendix One - Resources and contacts' for a COPD self-management plan.

Psychosocial functioning

Psychosocial support is essential and can be provided through local Asthma Societies and some COPD groups.

Carers (often family members) are critical to the care of patients with COPD. This often results in physical and emotional health problems for the carers and may threaten their relationships.

COPD impacts greatly on a person's ability to function in the community. Depression, anxiety and panic are frequent complications. Patients with COPD may fear that the end of their lives will be miserable and they will eventually suffocate to death.

Conventional pharmacotherapy is often useful for depression and choice of therapy should be made on the usual clinical criteria, but patients need to be carefully monitored for side effects such as aggravation of sleep disturbance or interference with respiratory control. Cognitive behaviour therapy can be beneficial.

X eXacerbations - manage appropriately

Management of exacerbations

An exacerbation of COPD is a sustained worsening of symptoms from the usual stable state and is acute in onset. Frequently reported symptoms are worsening breathlessness, cough, increased sputum production and change in sputum colour (NICE, 2004).

A wide range of other comorbidities may confuse the diagnosis of an exacerbation of COPD as they produce similar symptoms. They include:

- Pneumonia
- Pneumothorax
- Left ventricular failure/pulmonary oedema
- Pulmonary embolus
- Lung cancer
- Upper airway obstruction
- Pleural effusion
- Recurrent aspiration
- Arrhythmias

Exacerbations can usually be managed at home unless the exacerbation is severe or of rapid onset, the diagnosis is in doubt, oxygen is needed, or there are other medical or psychosocial reasons for admission.

Treatment options for an exacerbation of COPD managed at home include:

- For most patients:
 - o Increase dose and frequency of inhaled short-acting bronchodilators via spacer.
 - o Prednisone 40mg daily by mouth for 7-14 days.
- For patients with increased or more purulent sputum, in addition to above:
 - o Antibiotics: starting usually with amoxicillin 500mg TDS for 7-10 days, **or**, doxycycline 100mg daily for 7-10 days.
 - Macrolide antibiotics are often ineffective in bacterial exacerbations of COPD and may also increase theophylline levels.

The earlier an exacerbation is treated the better the results and so most patients who get exacerbations will benefit from having a supply of prednisone and a suitable antibiotic at home.

Review of drug use in COPD

Drugs currently available for treatment of COPD can reduce or eliminate symptoms, increase exercise capacity and lung function, reduce the number and severity of exacerbations and improve quality of life. However, there are no drug treatments currently available that modify the rate of decline in lung function. The improvement in lung function often seen with brief drug treatment does not necessarily predict other clinically related outcomes (ATC, 2004). The combination of different drug treatments can produce a greater change in spirometry and symptoms than single agents given alone.

Antibiotics

The benefits of long term antibiotic use do not outweigh the harms of treatment and the risk of promoting antibiotic resistance (Clinical Evidence, 2004).

Antibiotics should be used in exacerbations when patients have an increase in cough, dyspnoea, sputum production or purulence (NZGG, 2002).

Inhaled bronchodilators

Short acting beta-2agonists (salbutamol, terbutaline)

These act directly on smooth muscle to cause bronchodilation. They also appear to reduce hyperinflation by reducing air-trapping in the lungs. This may explain why some patients benefit from them without an improvement in spirometry or PEFr. Effects last for about four hours and time to peak response is slower than in asthma. There is evidence that they increase FEV₁ and reduce breathlessness, dyspnoea and fatigue. They can be used intermittently or regularly. There is no evidence that regular use of short acting beta-2 agonists adversely affects survival in patients with COPD (Prodigy, 2004).

Short acting anticholinergics (ipratropium)

Ipratropium blocks cholinergic nerves therefore blocking bronchoconstrictor effects. Mucus secretion is reduced by blockade of muscarinic receptors. They may also reduce hyperinflation providing similar benefits to beta-2agonists. Ipratropium increases FEV₁ but like short acting beta-2 agonists it has no effect on the rate of progression of COPD. They can be used intermittently or regularly.

Short acting beta-2 agonists or ipratropium

In practice there is little to choose between ipratropium and a short acting beta-2 agonist and either can be used as initial therapy. Ipratropium has a slower onset of action than beta-2 agonists and is less suitable for as required use (Prodigy, 2004).

There have been no long term comparisons of short acting inhaled anticholinergics and short acting inhaled beta-2 agonists. One non-systematic review found that short term (3 months) regular treatment with ipratropium is more effective at improving FEV₁ than regular treatment with short acting beta-2 agonist bronchodilators (Rennard, 1996).

The response to treatment should be assessed over at least four weeks. Therapy should be continued if there is documented improvement in lung function, symptoms of breathlessness, objective measures of breathlessness (MRC scale) or exercise capacity. If the response to a short acting beta-2 agonist is unsatisfactory a short acting anticholinergic should be trialed and vice-versa (NZGG, 2002).

Long acting inhaled anticholinergics (tiotropium)

Tiotropium has a similar mode of action to ipratropium but has a duration of action >24 hours and is administered once daily. It increases FEV₁ compared with placebo and it improves dyspnoea, and reduces exacerbation rates compared with placebo and regular ipratropium (NZGG, 2002; Prodigy, 2004).

Tiotropium has been compared with long acting beta-2 agonists in three randomised controlled trials. Two trials found that tiotropium improved FEV₁, and health related quality of life compared with salmeterol over six months. However, another trial found no significant difference between the same treatments in health related quality of life or exacerbation rates at six months (Clinical Evidence, 2004).

Tiotropium is funded in New Zealand for the treatment of severe COPD on completion of a Special Authority Subsidy form.

Long acting beta-2 agonists (salmeterol, eformoterol)

The bronchodilator effects are similar to the short acting drugs but the duration of action is around 12 hours. These drugs have been found to reduce dyspnoea and symptom scores for COPD when compared with placebo (Prodigy, 2004).

Long acting bronchodilators provide sustained relief of symptoms in moderate to severe COPD (NZGG, 2002). The evidence for effects on lung function and exacerbation rates is conflicting. Some trials have shown significant benefit whereas others have found no effect (Clinical Evidence, 2004; Prodigy, 2004).

Long acting beta-2 agonists are not currently funded in New Zealand for use in COPD.

Inhaled corticosteroids

Inhaled corticosteroids are not first line treatments for COPD. The inflammation that occurs in COPD is different to that seen in asthma and does not generally respond to inhaled corticosteroids. They have no effect on symptom scores or rate of decline in FEV₁. They reduce exacerbation rates in severe COPD (FEV₁ < 50%) but have no effect on exacerbation rates in mild COPD (Prodigy, 2004). Short term (10 days to 10 weeks) randomised controlled trials of inhaled corticosteroids in patients with COPD have found no evidence of superiority over placebo at improving FEV₁. A systematic review of long term trials (3 trials, 197 patients, treated for 2-2.5 years) also found no improvement in FEV₁ compared with placebo.

A trial of inhaled corticosteroids is recommended in patients with an FEV₁ less than or equal to 50% of predicted, who have had two or more exacerbations requiring treatment with oral corticosteroids or antibiotics in a 12 month period (NICE, 2005). They could also be trialed in patients with moderate or severe COPD with monitoring of objective measures of response. If there is no response they should be discontinued (NZGG). There is a possibility of exacerbation following withdrawal and re-instituting therapy may be necessary (Jarad, 1999).

Inhaled corticosteroid and long acting beta-2 agonist combination

Recent trials (Clinical Evidence, 2004) have demonstrated some benefits from the combined administration of inhaled corticosteroids and long acting beta-2 agonists. When the combination was compared with placebo there was a reduced number of exacerbations, improved lung function and improved health related quality of life scores. Furthermore the combination was more effective than for either drug used alone.

However these trials have been performed in patients with moderate to severe disease (FEV₁ <50%) and therefore the results cannot be generalised to patients with less severe COPD.

The Global Initiative for Chronic Obstructive Lung Disease (GOLD, 2003) advocates that the combination should only be used in patients with an FEV₁ of less than 50% predicted and frequent exacerbations (at least 3 in the last 3 years) (GOLD, 2003).

The combinations are not funded for COPD in New Zealand

Oral corticosteroids

Maintenance use of oral corticosteroids is not usually recommended but they may be required in patients with advanced COPD, especially if withdrawal is difficult following an exacerbation. The dose of corticosteroid should be kept as low as possible and patients should be monitored for the development of osteoporosis and diabetes. Long term use of oral corticosteroids is not recommended.

Oral corticosteroid reversibility tests do not predict if the patient will respond to inhaled corticosteroids (NICE, 2004).

Theophylline

Some patients with severe COPD and disabling breathlessness may benefit from slow release theophylline added to existing therapy.

Theophylline is a bronchodilator and may have an anti-inflammatory effect and reduce muscle fatigue but the exact mechanism of action is unknown. Its use is limited by the potential for drug toxicity as the drug has a narrow therapeutic index requiring plasma concentration monitoring. Levels should be at the lower end of the therapeutic range, that is 40-60 micromol per litre.

Significant adverse drug reactions and drug interactions are associated with theophylline use. Hypokalaemia may be potentiated by combined use with beta-2 agonists and is potentially hazardous. The elderly are at greater risk of adverse effects.

Theophylline increases FEV₁ and FVC compared with placebo but there is no significant improvement in wheeze, dyspnoea, walking distance, use of rescue medication or exacerbations (Prodigy, 2004).

Mucolytics

A trial of a mucolytic agent can be considered in patients who have difficulty in expectorating sputum.

Mucolytics (e.g. Bromhexine) have been available for many years and most of the evidence supporting their use comes from trials conducted in patients with chronic bronchitis. A systematic review of 22 trials (20 chronic bronchitis and not further defined, 2 COPD) found that 3-6 months treatment with mucolytics was more effective than placebo in reducing the number of exacerbations. It is not clear if these results can be extrapolated to patients with a confirmed diagnosis of COPD, but a trial of a mucolytic agent can be considered in patients who have difficulty in expectorating sputum. If symptoms improve the mucolytic should be continued (NICE, 2004).

Appendix One - Resources and contacts

Asthma and Respiratory Foundation of New Zealand

Email: arf@asthmanz.co.nz

Web: www.asthmanz.co.nz

- Patient support
- COPD Handbook
- COPD self-management plans
- Diagnosis & Treatment of COPD flow chart
- Spirometry services handbook

The National Heart Foundation of New Zealand

Email: info@nhf.org.nz

Web: www.nhf.org.nz

- Free smoking cessation practitioner training

Goodfellow Unit at the University of Auckland

Web: www.health.auckland.ac.nz/goodfellow/contracts/tads.html

- Tobacco, Alcohol and Other Drugs Early Intervention Training Programme (TADS)

Aukati Kai Paipa and other Māori cessation programmes

Web: www.auahikore.co.nz/contacts/aukati.htm

- Provide kapupapa Maori smoking cessation programmes

Ministry of Health

Web: www.healthed.govt.nz

Locally: local Public Health provider

- Providers of The Quit Book

Quitline

Web: www.quit.org.nz

Email: quit@quit.org.nz

- Quitcards supplied to providers registered with the programme
- Quitline (0800 778 778)

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