GUIDELINE

GUIDELINES FOR THE MANAGEMENT OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Working Group of the South African Pulmonology Society

Objective. This guideline has been developed in order to optimise the management of patients with chronic obstructive pulmonary disease (COPD) at all levels of the health care system in South Africa. It contains an action plan for early recognition and appropriate treatment of this common condition.

Options. Treatment regimens are recommended for patients with mild (stage I), moderate (stage II) and severe (stage III) disease.

Outcomes. Optimal management of patients with COPD may achieve a reduction in breathlessness, improved quality of life, prevention of complications and limitation of disease progression.

Evidence. The Working Group comprised mainly pulmonologists, but included an anaesthetist, a pharmacologist and a physiotherapist. Detailed literature review with particular attention to similar guideline documents from Europe and the USA was performed before the meeting.

Recommendations. Steps in the management of patients with COPD include early recognition of the disease, smoking cessation, treatment of airflow obstruction with appropriate drugs (singly or in combination), education and pulmonary rehabilitation, and limitation of disease progression and complications. Detailed recommendations are made with regard to the use and interpretation of a trial of oral corticosteroid therapy. Indications for hospitalisation, intensive care unit admission and ventilatory support are provided.

Validation. This guideline is similar to those recommended by other groups outside South Africa. It was developed by a working group of the South African Pulmonology Society and is endorsed by the Medical Association of South Africa.

Sponsors. The meeting of the Working Group was sponsored by Boehringer Ingelheim. This sponsorship did not influence the activities of the Group.

S Afr Med J 1988; **88**: 999-1010.

Working Group of the South African Pulmonology Society:
E D Bateman (Chairman), I Abdullah, G Ainslie, P Bardin, J Blott, A Coetzee, D Cohen, C Feldman, M Greenblatt, J Jansen, J Joubert, U Lalloo, Y Moodley, R Morar, O Mzileni, J O'Brien, W Otto, D Pansegrouw, M Plit (Convenor), R Raine, M Kamdar, G Richards, G Tintinger, B van der Wal, E van Schalkwyk, S Visser, G Walzl, M Wong

Report compiled by: **J O'Brien, C Feldman, E Bateman, M Plit**

Contact person: **Dr J A O'Brien, PO Box 16433, Vlaeberg, 8018**

Guideline sponsors: **The meeting of the Working Group of the South African Pulmonology Society was sponsored by Boehringer Ingelheim (Pty) Ltd**

DEFINITIONS

Chronic obstructive pulmonary disease (COPD) includes the conditions known as chronic bronchitis and emphysema. It is a disease state resulting predominantly from smoking tobacco and is characterised by airflow obstruction which is generally progressive. It may be accompanied by hyperreactive airways, and the airway obstruction may be partially reversible.

Chronic bronchitis refers to the presence of a chronic productive cough for at least 3 months of the year in 2 or more successive years in the absence of other recognised causes of chronic cough.

Emphysema is a pathological diagnosis describing permanent abnormal enlargement of the airspaces distal to the terminal bronchioles, accompanied by destruction of alveolar walls.

EPIDEMIOLOGY OF COPD

COPD is often diagnosed late because patients lack symptoms in early stages of the disease despite the presence of moderate decreases in pulmonary function. The primary risk factor for COPD is cigarette smoking. However, in South Africa tuberculosis and exposure to industrial and mining dust probably constitute important additional causes of COPD. Deficiency of alpha-1-antitrypsin (alpha-1-protease inhibitor) is a rare cause of disease. Patients with COPD have a more rapid age-related decline in forced expiratory volume in 1 second (FEV_1) than normal subjects. This accelerated decline can be slowed by smoking cessation. It is not clear whether recurrent infectious exacerbations contribute to long-term worsening of COPD. No reliable morbidity and mortality figures exist for South Africa, but overseas studies have shown that mortality is related to severity of disease. For example, an FEV_1 of < 35% predicted carries a mortality of approximately 30% at 1 year.

AIMS OF THIS GUIDELINE

The purpose of this Guideline is to improve patient care at all levels of the health system in South Africa (Table I), and it contains an action plan for the recognition and appropriate treatment of this common condition. It is not intended to be prescriptive.

Table I. Goals of management

1. Recognition of disease (early diagnosis and staging)
2. Smoking cessation (secondary prevention)
3. Improvement of breathlessness (treatment of airflow obstruction)
4. Pulmonary rehabilitation and education (improving quality of life)
5. Prevention and treatment of complications and limitation of disease progression

1. RECOGNITION OF DISEASE (EARLY DIAGNOSIS AND STAGING)

Consider this diagnosis in the presence of chronic dyspnoea and/or chronic cough with sputum production, and a long history of smoking, usually more than 15 pack-years (1 pack-year equals 20 cigarettes per day for one year).

To ensure that the correct treatment plan is followed, it is important to differentiate asthma from COPD (Table II).

Table II. Distinguishing features of COPD and asthma

Features suggesting COPD

- Persistent unremitting dyspnoea and productive cough despite treatment
- A long history of smoking
- Slow progression
- Hyperinflation and abnormal spirometry while in a stable state
- Cyanosis

Features suggesting asthma:

- Young age of onset
- Presence of atopy and/or allergic rhinitis
- Diurnal and day-to-day variation and seasonal variability
- Marked improvement after bronchodilator and/or 2-week trial of systemic steroids

Also note:

- Asthma and COPD may coexist, and distinguishing them may be difficult
- Breathlessness occurs late in COPD
- Asthmatics who smoke may have an accelerated decline in lung function
- Industrial exposure (e.g. to silica dust) and previous tuberculosis may be associated with the development of COPD

1.1 Spirometry

Spirometry is essential for the detection, diagnosis, assessment and management of patients with COPD and should be performed by adequately trained persons using a spirometer of the approved standard and calibrated regularly.

Peak expiratory flow rate measurements, while helpful in asthma, are unsuitable for assessment of COPD.

Spirometric indices include the forced vital capacity (FVC) and FEV_1. Spirometry permits the following:

Detection of airflow obstruction

All patients with symptomatic COPD have airflow obstruction with a reduced FEV_1.

To diagnose airflow obstruction, the FEV_1 is expressed as a percentage of the FVC. A value of less than 70% indicates disease.

To assess the severity of airflow obstruction, FEV_1 is expressed as a percentage of predicted values (in South Africa, predicted values for the black population are 10% lower).

Absence of airflow obstruction should alert to the possibility of an alternative diagnosis.

Assessment of degree of reversibility

Spirometry should be performed before and 20 minutes after two puffs of a short-acting beta-2-agonist bronchodilator. An improvement in FEV_1 of 15% and > 200 ml signifies a significant reversible component and should alert to the possibility of asthma.

Monitoring of a trial of systemic corticosteroids

For significant persistent breathlessness a trial of oral corticosteroids is given (Table III). *This must be monitored with spirometry* (Table IV).

Table III. Corticosteroid trial

Indicated in the assessment of all stage II and III patients
May also be used to assess reversibility in stage I patients

- Prednisone 40 mg daily for 14 days
- Objective assessment of response must involve:
  Detailed record of improvement in effort tolerance.
  Spirometry:
  Improvement of FEV_1 of > 15% and > 200 ml — patient has a significant reversible component. Treat as for asthma
  Improvement in FEV_1 by < 10% and significant symptomatic improvement — if stage III consider low-dose oral steroids* and optimise bronchodilators
  No objective response — optimise bronchodilators and other supportive therapy, stop corticosteroids

* Consider serious adverse effects of oral corticosteroids .

Assessment of the severity of disease

Spirometric staging is used to assess the severity of disease (Table IV).

Table IV. Spirometric staging of severity of COPD (performed while clinically stable)

	FEV_1 (% pred)	Symptoms	Comments
Stage I (mild)	≥ 50%	Mild effort-related dyspnoea	Good quality of life Little morbidity No hypoxaemia Common
Stage II (moderate)	≥ 35% ≤ 49%	Continuous dyspnoea which interferes with lifestyle.	Considerable morbidity
Stage III (severe)	< 35%	Dyspnoea limits activities of daily living	Respiratory failure and/or cor pulmonale common High mortality

Patients with symptoms and corresponding spirometry compatible with stages II and III should preferably be referred for assessment by a pulmonologist.

Monitoring disease progression

COPD progresses slowly over years. More rapid deterioration indicates an alternative diagnosis, e.g. asthma, cardiac failure, pneumothorax.

1.2 Chest radiograph

The chest radiograph on its own is not diagnostic, and a normal chest radiograph does not exclude COPD. It is not essential in all cases but may be useful for detecting additional disease. The presence of bullae and/or scarring from previous tuberculosis may indicate irreversible airway obstruction.

2. SMOKING CESSATION (SECONDARY PREVENTION)

- This is the only measure that slows progression of COPD.
- Limited improvement occurs in many cases.
- Smoking cessation strategies must be implemented (see supplementary note).

3. IMPROVEMENT OF BREATHLESSNESS (TREATMENT OF AIRFLOW OBSTRUCTION)

For summary see Fig. 1.

3.1 Stage I

If the patient is breathless, treat with one of the following bronchodilators (see supplementary note).

Beta-2-agonist metered dose inhaler (MDI)
(2 puffs approximately 6-hourly as needed)
or
Ipratropium MDI
(2 puffs approximately 6-hourly as needed)
or
Combination MDI of ipratropium and beta-2-agonist
(2 puffs approximately 6-hourly as needed)
or

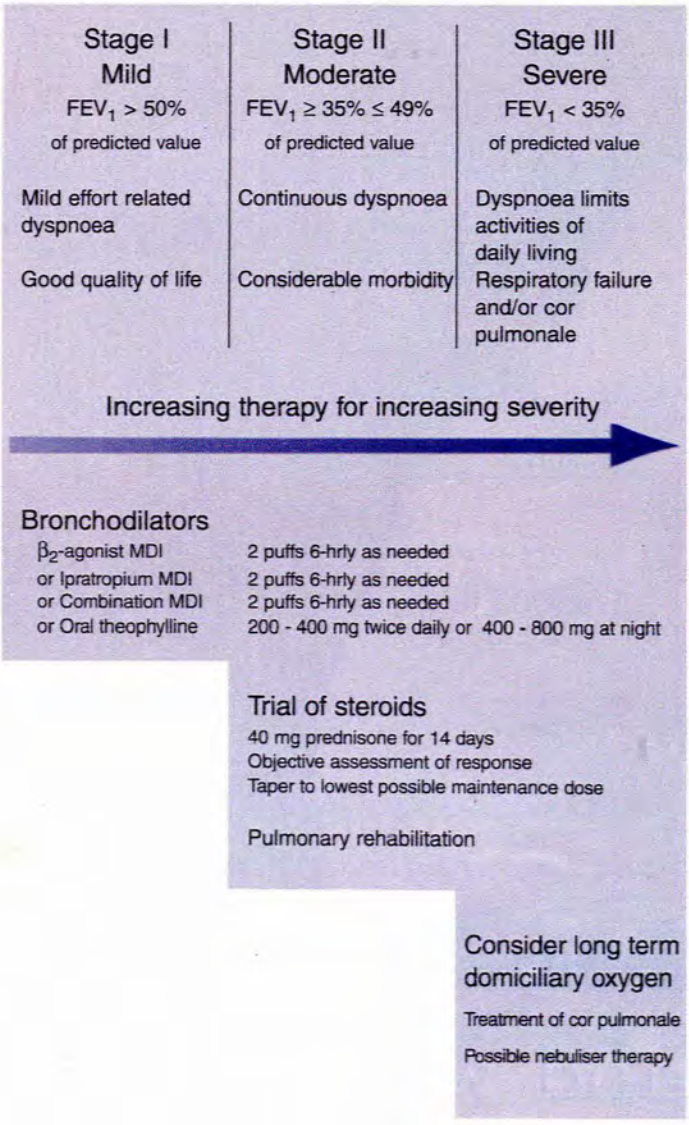

Fig. 1. Treatment guidelines for COPD (South African Pulmonology Society Working Group).

Regular oral SR theophylline
(200 - 400 mg twice daily or 400 - 800 mg at night).

3.2 Stage II and stage III

A trial of corticosteroids

A trial of corticosteroids forms part of the diagnostic assessment and is an indicator of likely responsiveness to therapy. It establishes a target lung function for bronchodilator treatment and should be performed unless a medical contraindication exists. Patients should be in a stable phase.

A short trial of corticosteroids will not reactivate tuberculosis. When active tuberculosis is suspected and in cases where long-term steroid therapy is planned, an initial chest radiograph is advised.

Bronchodilators

Regular beta-2-agonist and ipratropium MDI or equivalent (usually 2 (up to 6 in severe cases) puffs approximately 6-hourly as needed)
with or without
Regular oral SR theophylline
(200 - 400 mg twice daily or 400 - 800 mg at night).

Long-term corticosteroids

Chronic oral corticosteroids improve symptoms and lung function in approximately 20% of patients, but because of side-effects these should be used with caution. They should only be used when objective evidence of improvement in stable patients has been obtained. Regular attempts to reduce the steroid dose should be made.

Although there is evidence of some airway inflammation in COPD, the benefits of long-term preventive inhaled corticosteroid treatment, recommended for asthma, have not been confirmed. However, patients with a significant reversible component who respond objectively to oral corticosteroids may be given inhaled corticosteroids as a means of avoiding or reducing the need for oral steroids.

Mucolytics and mucokinetic agents

Expectoration of tenacious sputum is a distressing symptom. Unfortunately mucolytics, mucokinetic drugs, cough syrups and acetylcysteine (oral and inhaled) have not been shown to be effective and are not recommended.

Chest physiotherapy

An ineffective cough may be improved by instruction in the 'huff technique' of coughing and active cycle of breathing. Percussion and vibration therapy do not form part of routine management in stable patients.

The physiotherapist has an important role in directing the conditioning (exercise) programme and in advising on breathing and coughing techniques.

Other anti-asthma drugs

Other anti-asthma drugs, e.g. sodium cromoglycate, ketotifen and nedocromil sodium, are not useful in COPD.

Venesection

Increased haematocrit causes aggravation of cardiac failure, increased ventilation/perfusion abnormality and an increased incidence of thrombotic episodes. When the haematocrit is > 0.55 consider repeated therapeutic venesection.

Other techniques

Bronchoscopy and pulmonary lavage has no role.

4. PULMONARY REHABILITATION AND EDUCATION (IMPROVING QUALITY OF LIFE)

Rehabilitation programmes improve exercise tolerance and quality of life. Rehabilitation involves a multidisciplinary programme of physiotherapy, muscle training, nutritional support, psychotherapy and education. Comprehensive programmes are desirable and should be offered in specialised centres.

4.1 Education

Education involves the following advice (preferably written):

- Benefits and techniques of smoking cessation
- Pathophysiology of disease
- Prognosis
- Drug treatment and side-effects of drugs
- Crisis plan for attacks of severe breathlessness or infection — who to contact and how to cope
- Physiotherapy techniques
- Goals of exercise programmes
- Use of oxygen.

4.2 Physical conditioning

At a simple level patients should be given a graded programme of upper and lower limb exercise, e.g. free-range walking. Cycling and treadmill walking are useful alternatives. More sophisticated programmes have limited benefit. Hypoxic patients should be exercised with caution.

Breathing exercises, e.g. pursed lip breathing to improve respiratory flows, 'diaphragmatic breathing' and relaxation techniques, may be of benefit. Energy conservation measures for the severely breathless include synchronised breathing, avoidance of breath-holding, arm-support during activities such as shaving, devices to aid towelling and bathing, etc.

4.3 Psychological support

This involves group and family support and advice to the family from the practitioner and other care-givers.

4.4 Nutrition

Obesity and loss of body mass are both common features of COPD. Obese patients should be advised to lose weight. Under-nutrition is associated with respiratory muscle

dysfunction and increased mortality. Weight gain in advanced COPD is generally difficult to achieve. Simple advice to take frequent small meals and not have large meals shortly before retiring may improve nutrition and limit the dyspnoea associated with eating.

5. PREVENTION AND TREATMENT OF COMPLICATIONS AND LIMITATION OF DISEASE PROGRESSION

5.1 Treatment of right ventricular failure

- Administer oxygen if hypoxaemic.
- Diuretics (e.g. hydrochlorothiazide 25 mg or equivalent). Avoid large decreases in preload which may precipitate hypotension.
- Cardiac glycosides are to be avoided except in the presence of atrial fibrillation and/or left ventricular dysfunction/failure.
- ACE inhibitors and calcium antagonists are not indicated in cor pulmonale or right ventricular failure.

5.2 Infections in COPD

Prevention

Southern hemisphere influenza vaccination annually in March is advised.

Treatment

In the presence of purulent sputum, a course of antibiotics such as amoxycillin, or one of the tetracycline derivatives (e.g. doxycycline), is recommended for a minimum of 10 - 14 days. Alternatives include new macrolide/azalide agents, amoxycillin/clavulanate and second-generation cephalosporins. Quinolones may also be used as second-line agents, especially in severely ill patients, particularly if guided by sputum culture.

5.3 Treatment of hypoxaemia — oxygen therapy

Long-term domiciliary oxygen therapy (LTDOT)

This form of therapy, when given for the correct indications with correction of hypoxaemia, results in reduction of complications of respiratory failure and significant improvement in survival of patients with chronic respiratory failure.

Indications for LTDOT:

- Non-smokers with stable, severe chronic obstructive pulmonary disease (FEV_1 < 1.5 litres)
- Arterial hypoxaemia (partial arterial oxygen pressure (PaO_2) < 7.3 kPa or 55 mmHg)
- With or without hypercapnia (partial arterial carbon dioxide pressure ($PaCO_2$) > 6.0 kPa or 45 mmHg)
- With or without oedema.

The disease must be stable and patients must be on appropriate drug treatment before assessing hypoxaemia, as blood gases may continue to improve for up to 3 months after an acute exacerbation. Both arterial hypoxaemia and spirometric values must be tested on two occasions at least 1 month and preferably 3 months apart before prescribing LTDOT.

Continued smoking reduces the efficacy of treatment and is a contraindication to oxygen therapy. Blood levels of carboxyhaemoglobin above 3% suggest continued smoking.

Severe airflow obstruction (low FEV_1) must be confirmed since LTDOT is not of proven benefit in other forms of disease.

Oedema indicates cor pulmonale and a worse prognosis. LTDOT has a significant benefit in this group.

In patients with hypercapnia, oxygen therapy must be carefully controlled and arterial $PaCO_2$ should be assessed to ensure that carbon dioxide retention does not occur.

Hypoxaemia is assessed with the patient at rest. Patients in whom falls in PaO_2 occur only during exercise and sleep do not benefit from LTDOT.

Other components of rehabilitation must be provided.

The patient (and family) must understand the nature and goals of therapy and be aware that it is not intended for relief of dyspnoea alone.

Regular follow-up by a suitably experienced physician and ready access to technical advice, either through a private contractor or a hospital department, must be available. Assessment of compliance is essential.

The oxygen prescription. Oxygen is administered by facemask or cannulae for a total of at least 15 hours during a 24-hour period. A flow rate of 1 - 2 l/min is used, the rate being determined in each case by arterial blood gas (or oximetry measurements after initial blood gas analysis) determinations.

Oxygen can be delivered by oxygen concentrators or by cylinders. Concentrators are more convenient and cost-effective.

Palliative symptomatic oxygen therapy

When given for short periods to relieve breathlessness in hypoxic patients, this does not influence the natural progression of the disease and is therefore not routinely recommended.

5.4 Evaluation and treatment of acute exacerbations of COPD

Evaluation

Clinical assessment. Determine symptom changes from baseline status, including sputum volume and character, duration and progression of symptoms, dyspnoea severity, exercise limitation and effect on activities of daily living.

Look for evidence of respiratory distress, bronchospasm, cor pulmonale and right ventricular failure, pneumonia, haemodynamic instability and altered mentation.

Investigations. Arterial blood gas or pulse oximetry (if blood gas is not available), chest radiograph, ECG and theophylline level (if the patient has been on theophylline).

Treatment

Bronchodilators: (*i*) nebulisation with a combination of ipratropium bromide and beta-2-agonist is usually indicated as often as every 30 - 60 minutes in severe cases. Early conversion to MDI is desirable. Alternatively multiple actuations of an MDI with a spacer may be as effective; (*ii*) theophyllines — should there be inadequate sustained response to the above treatment, intravenous aminophylline may be used (Table V).

Table V. Theophylline guidelines

- Loading dose IV of 6 mg/kg if patient not on oral theophylline
- Maintenance dose of 0.6 mg/kg/hour given as an infusion
- Blood level monitoring and dose reduction if symptoms of toxicity develop
- Reduce dose in elderly and those with cardiac failure

Anti-inflammatory therapy. Corticosteroids should be given, preferably orally. A once-daily dose of 40 mg prednisone is given and continued for 10 - 14 days unless the condition fails to resolve. Tapering is not required.

An equivalent dose of an intravenous steroid may be given if the patient is unable to take oral medication.

Antibiotics. These should be prescribed when there is evidence of infection or increasing breathlessness considered to be due to infection. Empiric antibiotic therapy is used and includes oral amoxycillin, doxycycline or co-trimoxazole. Alternatives such as amoxycillin/clavulanate, cefuroxime, macrolides/azalides or quinolones may be used, particularly in areas of high levels of resistant *Haemophilus influenzae*. Intravenous administration is preferred for severe illness and pneumonia on chest radiograph.

The duration of treatment should be 10 - 14 days for severely ill patients or for persistent infections.

Postural drainage and chest percussion are of limited benefit except in patients with excessive secretions.

Oxygen therapy. Oxygen should be started at 24% or 1 - 2 l/min by nasal cannulae. Increases should be gradual to avoid carbon dioxide narcosis. This should be guided by blood gas analysis or by level of consciousness if blood gases are not available. The aim should be to maintain the saturation above 90%.

Treatment of cardiac failure. Right-sided cardiac failure is treated primarily with oxygen and diuretics. As in chronic right ventricular failure, there is no role for digoxin, calcium-channel blockers or ACE inhibitors in acute right heart failure. It may be necessary to use these agents if left ventricular failure coexists.

Heparin prophylaxis. To prevent deep-vein thrombosis, heparin should be given subcutaneously if there is no contraindication.

Miscellaneous: (*i*) there is no place for oral or inhaled mucolytics; (*ii*) coughing and forced expiratory manoeuvres aid in the clearance of secretions. Instruction in correct breathing patterns may relieve dyspnoea.

Indications for hospitalisation, ICU admission and ventilatory support are summarised in Table VI.

6. Additional considerations

6.1 Sleep in COPD

Sleep is associated with a decrease in arterial oxygen saturation (SaO_2) in most individuals, but this trend is more marked in COPD.

Significant night-time hypoxaemia cannot be predicted from measurement of daytime blood gas and pulmonary function tests, but if the daytime PaO_2 is ≥ 8 kPa, nocturnal SaO_2 need only be measured if unexplained respiratory failure, cor pulmonale or erythrocytosis is found.

Referral for a full sleep study (polysomnography) and specialised opinion should only be considered in the setting of suspected superimposed sleep-disordered breathing (i.e. in the presence of daytime hypersomnolence and other symptoms of sleep deprivation or a strong history of snoring or apnoeic events).

Long-term domiciliary oxygen therapy, prescribed for daytime hypoxaemia, must be used during sleep.

6.2 Surgery for emphysema

Giant bullectomy is occasionally indicated.

Lung volume reduction surgery (LVRS), bilateral non-anatomical excision of emphysematous lung tissue, might be a useful modality of therapy in an extremely small, highly selected group of patients. These patients must be on optimal medica. therapy, severely symptomatic with dyspnoea, and have evidence of marked air trapping without bullous disease. The patient should be referred to a pulmonologist who is participating in a multidisciplinary LVRS programme for evaluation.

6.3 COPD and surgery

Patients with COPD are at higher risk during anaesthesia and surgery. The risk depends on the severity of the lung disease and the nature of the proposed surgery.

All patients require pre-operative evaluation, including spirometry. For lung resection and upper abdominal and thoracic surgery, the minimum evaluation should include spirometry and arterial blood gas analysis.

Patients with continuous dyspnoea interfering with lifestyle (stages II and III) should undergo these investigations before any anaesthetic.

The anaesthetic should be performed by someone

Table VI. Indications for hospitalisation, ICU admission and ventilatory support in COPD

Hospitalisation	ICU admission	Ventilatory support
1. Acute exacerbation characterised by increased dyspnoea, cough or sputum production, plus one or more of the following: •Sustained failure to improve on outpatient management • Inability to walk between rooms (where previously mobile) • Family and/or physician unable to manage the patient at home. • High-risk co-morbid condition, pulmonary (e.g pneumonia) or non-pulmonary • Prolonged, progressive worsening of symptoms before emergency visit • Altered mentation • Worsening hypoxaemia • New or worsening hypercapnia 2. New or worsening right-sided cardiac failure unresponsive to outpatient management.	The usual indication for admission is respiratory failure. However, it is frequently necessary to admit patients with other diseases because they have COPD. Admission is subject to: 1. Good functional ability prior to an exacerbation if prior functional ability was known (patient coped with activities of daily living). If not known, the patient should be given the benefit of the doubt. 2. Possible need for mechanical ventilation, i.e.: • PaO_2 < 6.7 kPa (50 mm Hg) • pH < 7.3 • Confusion 3. The presence of a reversible element to the disease. Examples would be: infection, bronchospasm, oxygen-induced carbon dioxide narcosis, sedative administration or other associated illnesses.	Patients with COPD often have hypercapnia, hypoxaemia or severe dyspnoea and these alone are not an indication for ventilatory support. Specific indicators may be one or a combination of (presuming the presence of reversible factors): 1. Hypoxaemia (PaO_2 < 50 mm Hg) despite supplemental oxygen 2. Exhaustion, confusion, coma 3. pH < 7.3 and declining (respiratory acidosis) 4. Respiratory or cardiac arrest 5. Inability to clear secretions Ventilatory support may consist of invasive (mechanical ventilation) or non-invasive (CPAP, BiPAP) techniques

Discharge plan
1. Education.
2. Consideration for domiciliary oxygen.
3. Provide written home management and treatment guideline.
4. Outpatient appointment arranged.
5. Rehabilitation plan.
6. Smoking cessation.
7. Psychosocial support.

experienced in dealing with the anaesthetic complications and problems arising in these patients.

SUPPLEMENTARY NOTES

Smoking cessation

The important benefits of smoking cessation are that it reduces the rate of decline of lung function to that of the normal ageing process. Blood transport of oxygen improves. An initial small increase in FEV_1 occurs in many patients. Although many interventions show impressive short-term results, long-term continued abstinence occurs in fewer than 30% of patients. Factors associated with poor success include slow reductions in cigarette consumption, multiple previous attempts to stop smoking, heavy smoking, and relapse within the first 2 weeks.

Features of a **successful programme** include: (*i*) advice and explanation by doctor and other caregivers/friends/family; (*ii*) repeated reinforcement and follow-up; (*iii*) a variety of forms of psychotherapy which may involve group, individual or behavioural methods; (*iv*) abrupt cessation (set quit date; 'cold turkey'); and (*v*) nicotine gum or patches.

Minor benefit: clonidine, buspirone.

No benefit: hypnosis, acupuncture.

Avoidance of occupational and atmospheric pollution is essential in alpha-1-antitrypsin deficiency.

Bronchodilators

Because a large proportion of COPD patients have a degree of reversible airways obstruction, bronchodilators effect some relief of symptoms and improved lung function occurs in most cases. The magnitude of the effect is less than in asthma. Bronchodilators do not alter the progressive decline in FEV_1.

Beta-2-agonists (the inhaled route is preferred), anticholinergics and theophyllines are effective. Individuals vary in their responsiveness to each and combinations may have additive effects, possibly because the mode of action of each differs.

Short-acting beta-2-agonists

These agents have a rapid onset of action and achieve similar effects to anticholinergics. They may be used regularly and as monotherapy. Examples include salbutamol, fenoterol, terbutaline.

Anticholinergics

Anticholinergic agents have a slower onset of action (± 40 minutes to peak effects) but are effective for longer (6 hours), improving symptoms, effort tolerance and airflow obstruction. They are more effective than beta-2-agonists in many patients and have fewer side-effects.

They do not reduce mucociliary clearance, or have significant urinary or pupillary effects, even in high doses and in the elderly. Ipratropium bromide is the only inhaled anticholinergic currently available.

Nebuliser treatment

This is an alternative for stage III patients with poor inhalation technique and/or acute dyspnoea.

- Nebulised ipratropium plus beta-2-agonist can be used up to 3 or more times daily.
- While there are distinct advantages, nebulisers tend to be overused.
- Patients requiring nebuliser therapy should, where possible, have specialist assessment.

Theophyllines

Theophyllines have similar or less bronchodilator effect than beta-2-agonists but improve quality of life and have several additional therapeutic advantages. The oral route of administration is preferred by some patients and there is good compliance with the sustained-release preparations.

Limitations are toxicity (particularly in the elderly), drug interactions and variable metabolism.

Dosage: oral slow-release theophylline, 200 - 400 mg twice a day or 400 - 800 mg at night. Recommended doses should not be exceeded without monitoring blood levels.

A scheme for adjusting doses is as follows: (*i*) *increase* dose by one-third — smokers and patients on phenytoin therapy; (*ii*) *decrease* dose by one-third — patients with congestive cardiac failure, the elderly, patients with liver disease and those on most macrolide antibiotics, ciprofloxacin or cimetidine.

Combination tablets containing theophylline and other bronchodilators or sedatives are not recommended.

Long-acting beta-2-agonists

The use of long-acting beta-2-agonists in COPD is currently being evaluated. They may be useful, but at present they do not form part of routine management. A number of studies are underway which should help clarify their role and indicate which patients are most likely to benefit from their use. The currently available long-acting beta-2-agonists are salmeterol and formoterol.

Corticosteroids

Oral corticosteroids, used in the long term, may cause severe side-effects. The decision to use oral maintenance steroids should be made only if there is significant symptomatic and objective evidence of beneficial effect. The dose should be slowly reduced to the lowest that achieves adequate symptom control. The use of alternate-day dosing may reduce side-effects.

Patients should be monitored for common side-effects, which include weight gain, impaired glucose tolerance, skin fragility, hypertension and osteoporosis.

Inhaled corticosteroids may be used in steroid-responsive patients in order to try to reduce the systemic dose.

Spacer devices and delivery methods

The use of spacer devices may improve delivery of medication to the lower airways in patients with poor inhaler technique and poor lung function.

Some patients may achieve better results with dry powder devices. It is vital to ensure that the individual uses his inhaler device correctly.

Air travel

The risks of air treatment include:

- Worsening hypoxaemia at altitude.
  Most commercial aircraft are pressurised to an altitude of between 7 000 and 10 000 feet.
- Increased physical stress of travel (airport transfers, luggage, etc.).
- Risk of contracting an intercurrent infection with subsequent deterioration.
- Increased altitude at destination causing increased dyspnoea.

Patients need to balance the potential discomforts and risks with the intended objective of the trip.

The need for supplemental oxygen can be predicted by administering hypoxic air mixtures and measuring blood gases, or by use of regression equations. However, a simple approach is recommended:

- Patients who are on LTDOT, or are hypoxaemic at sea level, will need supplemental oxygen on the aircraft.
- Patients with limitation of activities of daily living should make provision for supplemental oxygen with the airline before the trip.
- Patients who have tolerated recent air travel well are likely to cope with a similar trip.
- Coexistent disease, particularly ischaemic heart disease, will make hypoxaemia more dangerous and more careful assessment is necessary.

Recommended reading

1. American Thoracic Society. Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease (COPD). *Am J Respir Crit Care Med* 1995; **152**: S77-S120.
2. Clark TJH, Pride N, Vermiere P, *et al*. Chronic obstructive pulmonary disease. Symposium.

Eur Respir J 1997; **7:** 45, 202-205.
3. COPD Guidelines Group of the Standard of Care Committee of the British Thoracic Society. BTS Guidelines for the management of chronic obstructive pulmonary disease. *Thorax* 1997; 52: suppl 5, S1-S28.
4. Niederman MS, ed. Mechanisms and management of COPD. *Chest* 1998; **113:** 233S-287S.
5. Siafakas NM, Vermiere P, Pride NB, *et al.* Optimal assessment and management of chronic obstructive pulmonary disease (COPD). European Respiratory Society Consensus Statement. *Eur Respir J* 1995; **8:** 1398-1420.

ANNEXURE A. METHODOLOGY OF THE WORKING GROUP

The South African Pulmonology Society has long recognised the need for a document to guide physicians and caregivers in the assessment and management of the common disease, chronic obstructive pulmonary disease. The Society has experience in formulating guidelines, and employed the following process.

A Working Group consisting of pulmonologists from seven South African medical schools and from private practice, a physiotherapist, a pharmacologist and an anaesthetist was formed. Each university was invited to send an additional two observers.

Prior to the first meeting of the Working Group the participants were asked to prepare reviews of selected topics in COPD, based on the relevant international literature. These reviews were distributed to the participants before the meeting.

The Group met on the weekend of 16 March 1997. The meeting was sponsored by Boehringer Ingelheim (Pty) Ltd. Company representatives provided logistic support, but did not take part in the discussions and were not involved in the preparation of the guideline document.

Important general issues such as definitions and classification of COPD were discussed in plenary sessions. The reviews of selected topics were then discussed in small groups in order to simplify and condense the document. The recommendations of the small groups were reviewed in a plenary session and the editorial committee was instructed to prepare the draft document. This was performed by the four-member editorial team by correspondence. A draft was prepared and sent to all members of the Working Group. After further comments on this draft were received, a second draft was prepared and discussed in detail at a further meeting of the Working Group held at the conference of the Pulmonology Society in Windhoek in September 1997.

Discussions at this meeting led to further changes to the document which was subsequently scrutinised and completed by the editorial committee prior to submission to the Guideline Committee of the Medical Association of South Africa in November 1997.

ANNEXURE B. MEMBERS OF THE WORKING GROUP AND AFFILIATIONS

E Bateman	University of Cape Town
I Abdullah	University of Cape Town
G Ainslie	University of Cape Town
P Bardin	University of Stellenbosch
J Blott	Private practice
A Coetzee	University of Stellenbosch
D Cohen	University of the Witwatersrand
C Feldman	University of the Witwatersrand
M Greenblatt	Private practice
J Jansen	University of Stellenboch
J Joubert	University of Stellenbosch
U Lalloo	University of Natal
Y Moodley	University of Natal
R Morar	University of the Witwatersrand
O Mzileni	MEDUNSA
J O'Brien	Private practice
W Otto	University of the Orange Free State
D Pansegrouw	University of the Orange Free State
M Plit	Private practice
R Raine	University of Cape Town
M Kamdar	University of Natal
G Richards	University of the Witwatersrand
G Tintinger	University of Pretoria
B van der Wal	University of Stellenbosch
E van Schalkwyk	University of Stellenbosch
S Visser	University of Pretoria
G Walzl	University of Stellenbosch
M Wong	University of the Witwatersrand