CLINICAL GUIDELINE

Guideline for the Management of Chronic Obstructive Pulmonary Disease (COPD): 2004 Revision

COPD Guideline Working Group of the South African Thoracic Society

Objective. To revise the South African Guideline for the Management of Chronic Obstructive Pulmonary Disease (COPD) in the light of new insights into the disease and the value of new treatment approaches and drugs. New aspects considered include:

- A growing awareness of the impact of COPD in South Africa, and the urgent need for prevention strategies
- The role of concurrent exposures to domestic and occupational atmospheric pollution, and previous lung infections including tuberculosis
- The need to consider as goals of treatment both prevention of exacerbations and improvement of quality of life (health status) of patients with COPD
- The development of both long-acting beta$_2$-agonist and anticholinergic drugs for use in COPD
- Emerging evidence on a limited role for inhaled corticosteroids in the treatment of COPD.

Recommendations. These include primary and secondary prevention; early diagnosis; staging of severity; assessment of reversibility with bronchodilator and, in some, responsiveness to corticosteroids; use of bronchodilators and other forms of treatment; rehabilitation; and treatment of complications. Advice is provided on the management of acute exacerbations, and the approach to air travel, prescribing long-term oxygen, and lung surgery including lung volume reduction surgery.

Prevention, both primary and secondary, remains the most cost-effective measure in the management of COPD, and deserves more emphasis, particularly on the part of health care professionals. Primary prevention involves reducing public exposure to cigarette and other forms of smoke, and reduction of atmospheric pollution, and secondary prevention limits exposure and resultant progression in those with established disease. Spirometry is essential for the diagnosis of COPD and in staging severity. In addition, a new classification of severity that considers other indices of functional impairment is provided. Treatment involves a progression from 'as-needed' bronchodilators, through the addition of other more effective bronchodilators, usually in combination, in more severe stages. The importance of assessing potential reversibility in every patient with persistent symptoms, and of the limited role of oral and inhaled corticosteroids (ICS), is emphasised. These approaches also reduce exacerbations and may result in cost savings and improved prognosis. A practical low-cost approach to rehabilitation is proposed.

Options. Treatment recommendations are based on the following: the recommendations of the Global Obstructive Lung Disease (GOLD) initiative, which provides an evidence-based comprehensive and up-to-date review of treatment options; independent evaluation of the level of evidence in support of some of the new treatment trends; and consideration of factors that influence COPD management in South Africa, including lung co-morbidity and drug availability and cost.

Outcomes. The use of bronchodilators is driven by the presence of symptoms, but regular assessment of benefit, based on objective criteria, is essential. Several forms of treatment reduce exacerbations, the most effective of these is smoking cessation.

Evidence. Working group of clinicians and clinical researchers following detailed literature review, particularly of studies performed in South Africa, and the GOLD guidelines.

Benefits, harms and costs. The guideline pays particular attention to cost-effectiveness in South Africa, and promotes the initial use of less costly options. It rejects empirical use of corticosteroids both oral and inhaled, and promotes smoking cessation, and selection of treatment based on objective evidence of benefit. It also rejects a nihilistic or punitive approach, even in those who are unable to break the smoking addiction.

Validation. The COPD Working Group comprised experienced pulmonologists representing all university departments in South Africa and some from private practice. All contributed to the development of the previous version of the South African guideline, and attend international meetings. One (JRJ) represents South Africa on the GOLD Guideline Executive.

Guideline sponsor. The meeting of the Working Group of the South African Thoracic Society was sponsored by an unrestricted educational grant from Boehringer Ingelheim (South Africa) (Pty) Ltd.

S Afr Med J 2004; **94:** 559-575.

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

Other members of the COPD Guideline Working Group of the South African Thoracic Society: **G M Ainslie, S Abdool-Gaffar, C Bolliger, A Foden, M Greenblatt, E Irusen, J Killian, U Lalloo, J Mpe, O Mzileni, W Otto, D Pansegrouw, R I Raine, G Richards, S Visser**

Corresponding author: **Professor E D Bateman, University of Cape Town Lung Institute, PO Box 34560, Groote Schuur, 7937, Cape Town. Tel: (021) 406-6901, fax (021) 406-6902, e-mail: ebateman@uctgsh1.uct.ac.za**

1. Management summary

Components of care

1. Recognition of disease (early diagnosis and staging). The patient presents with chronic breathlessness, wheezing and/or cough. The diagnosis is confirmed by spirometry: An FEV_1/FVC% (ratio of forced expiratory volume in 1 second to forced vital capacity) of less than 70% indicates airflow obstruction, and FEV_1 is an indicator of severity.

2. Smoking cessation to arrest disease progression.

3. Improvement of breathlessness through treatment of airflow obstruction, based on grading of severity.

4. Improvement of quality of life (pulmonary rehabilitation and education).

5. Prevention and treatment of exacerbations.

6. Prevention and treatment of complications.

Assessment of severity

	Stage 0	**Stage 1**	**Stage 2**	**Stage 3**
Grade of severity	Normal, but at risk	Mild	Moderate	Severe*
FEV_1 (% of predicted value)	> 80	79 - 60	59 - 40	< 40
Dyspnoea/ functional impairment	Normal exercise tolerance	Limits strenuous activity	Limits activities performed at 'normal' pace	Impairs activities of daily living, to virtual inactivity
6-Minute Walking Distance (m)	Normal (> 600)	< 600 - 200		< 200
Body mass index (kg/m²)	> 25	≤ 25 - 21		< 21

Management plan based on the grade of severity

Stage of COPD	**Prevention**	**Bronchodilators**	**Other drugs**	**Other measures**
Stage 0 $FEV_1 \geq 80\%$	• Education • Avoidance measures: smoking cessation			
Stage 1 FEV_1 60 - 79%	As above	On demand inhaled short-acting $beta_2$-agonist or anticholinergic broncho-dilator alone or combination inhaler or oral theophylline		
Stage 2 FEV_1 40 - 59%	As above	Symptom-driven regular use of inhaled short- or long-acting bronchodilators, alone or in combination with oral theophylline. Two or 3 might be needed with increasing severity. Long-acting $beta_2$-agonists include formoterol and salmeterol, and long-acting anticholinergic, tiotropium	A trial of oral or inhaled corticosteroid is indicated where FEV_1 is below 50% predicted. If objective benefit (in FEV_1 and effort tolerance) is found, or the patient has frequent exacerbations of COPD (3 or more per year) consider maintenance inhaled corticosteroids, or, rarely, low-dose oral corticosteroid	Prevention of exacerbations: • Influenza vaccination annually • Regular bronchodilators (with inhaled corticosteroids in a minority) Rehabilitation: • Education • Exercise programme • Psychological support • Nutrition
Stage 3 FEV_1 < 40%	As above	As above	As above	• Domiciliary oxygen • Treatment of cor pulmonale • Treat complications and co-morbidity

*Also severe if any of the following are present: repeated hospitalisation for exacerbations, co-morbidity, right heart failure, PaO_2 < 6.5kPa, age > 65 years, respiratory acidosis.

2. Introduction

Chronic obstructive pulmonary disease (COPD) is an important cause of death and disability in both developed and developing countries. It is becoming more common and accounts for significant and increasing utilisation of health care resources with attendant increases in health care expenditure. Cigarette smoking remains the major cause of COPD, but in Africa and Asia domestic biomass fuel use and tuberculosis are important additional causes.

3. Definitions

COPD is a disease state resulting predominantly from smoking tobacco, and is characterised by airflow obstruction, which is generally progressive and is only partially reversible.

The diagnosis of chronic bronchitis applies to patients who, in the absence of other recognised causes, e.g. bronchiectasis, have a chronic productive cough for at least 3 months of the year in 2 or more successive years. Emphysema is a pathological diagnosis describing permanent abnormal enlargement of the airspaces distal to the terminal bronchioles, accompanied by destruction of alveolar walls. Most patients with COPD have elements of both chronic bronchitis and emphysema in varying degrees, but some may have one without the other.

4. Epidemiology of COPD

COPD is often diagnosed late because patients lack symptoms in the 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, important contributory factors are tuberculosis, industrial and mining dust exposures, and domestic use of biomass fuels.[3,4] As a result of these additional factors, the prevalence of COPD is higher in poorer communities (Table I). It is not yet clear what impact HIV infection and its attendant pulmonary complications have on the pathogenesis and prevalence of COPD in South Africa.

Table I. Risk factors for the development of COPD in South Africa

Cigarette smoking
Pulmonary tuberculosis
Harmful exposures in mining and industry
Domestic use of biomass fuels
Smoking of marijuana and 'recreational drugs', e.g. methaqualone
Alpha-1 protease inhibitor deficiency (rare)
Childhood lung infections

Estimates of the global all-age incidence of COPD are that it affects 9/1 000 males and 7.3/1 000 females. It seldom presents clinically before the 5th decade, but incidence increases in successive decades and up to 50% of smokers over the age of 65 years are affected.[5] Incidence also varies widely between different regions and countries of the world, according to local cigarette smoking habits and domestic and socio-economic circumstances. It is more common in males than females, but morbidity among women has increased sharply in many countries, in parallel with an increase in the number of women smokers. Patients with COPD have a more rapid age-related decline in FEV_1 than normal subjects. This accelerated decline is slowed by smoking cessation. Recurrent infective exacerbations may also accelerate the rate of disease progression. Morbidity and mortality increases with severity of disease, age and co-morbidity.[6] The 2-year mortality is 20% in patients with an FEV_1 of 30 - 39%, and 40% in those with an FEV_1 of less than 30% of predicted. [7]

5. Objectives

The purpose of this guideline is to improve the care of patients with COPD in all sectors of care in South Africa. It includes an action plan for the recognition and appropriate treatment of this common condition, but is not intended to be prescriptive. The goals of management are listed below (Table II).

Table II. Goals of management of patients with COPD

1. Recognition of disease (early diagnosis and staging)
2. Smoking cessation to arrest disease progression
3. Improvement of breathlessness (treatment of airflow obstruction)
4. Improvement of quality of life (pulmonary rehabilitation and education)
5. Prevention and treatment of exacerbations
6. Prevention and treatment of complications

6. Goals of management

6.1. Recognition of disease (early diagnosis and staging of severity)

The diagnosis of COPD should be considered in any patient with chronic dyspnoea and/or chronic cough (with or without sputum production), a smoking history of more than 10 pack-years, and/or other risk factors for COPD (Table I), particularly if there is no other apparent cause for these symptoms (e.g. cardiac failure). One pack-year equals 20 cigarettes per day, one joint of cannabis per day, or 15 g of pipe tobacco per day, for 1 year. In South Africa, more than one risk factor is commonly found.

Correct diagnosis, and in particular, the differentiation of COPD from asthma is important to ensure correct treatment. Clinical features that assist in the diagnosis of each are outlined in Table III. The severity of COPD is defined on the basis of spirometry and various clinical features (Table IV).

6.1.1 Spirometry

Spirometry is essential for the detection, assessment and management of patients with COPD and must be performed by adequately trained persons using a spirometer of approved standard and quality that is calibrated regularly.[8] Measurements used in the diagnosis of COPD are FEV_1 after bronchodilator use and the ratio of FEV_1 to FVC (FEV_1/FVC%).

6.1.1.1 Detection of airflow obstruction

The presence of an FEV_1/FVC ratio of less than 70% confirms the presence of airflow obstruction. In these circumstances, the FEV_1 is usually reduced (less than 80% of predicted value), and is used as a measure of severity. Most patients with symptomatic COPD have a reduced FEV_1, but many patients with significantly reduced FEV_1 have no symptoms. An FEV_1 of < 80% of predicted after four inhalations of a short-acting $beta_2$-agonist in patients at risk, regardless of whether or not they have symptoms, suggests a diagnosis of COPD.

A slow or 'relaxed' vital capacity (SVC), that is, a vital capacity performed as a maximal but unhurried manoeuvre, frequently provides a larger value than the forced manoeuvre

Table III. Distinguishing features of COPD and asthma

Features suggesting a diagnosis of COPD
- Persistent unremitting dyspnoea, wheeze and productive cough despite treatment
- A long history of smoking
- Slow progression
- Hyperinflation and abnormal spirometry that persists during a stable phase of the disease
- Cyanosis

Features that suggest the presence of asthma
- Young age of onset
- Presence of atopy and/or allergic rhinitis
- Diurnal and day-to-day variation and seasonal variability
- Marked improvement after a bronchodilator and/or a 2-week trial of systemic steroids

Additional considerations in the diagnosis of asthma and COPD
- 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 are also associated with the development of fixed airflow obstruction

Table IV. Assessment of severity

	Stage 0	Stage 1	Stage 2	Stage 3
Grade of severity	Normal, but at risk	Mild	Moderate	Severe*
FEV_1 (% of predicted value)	> 80	79 - 60	59 - 40	< 40
Dysponea/functional impairment	Normal exercise tolerance	Limits strenuous activity	Limits activities performed at 'normal' pace	Impairs activities of daily living, to virtual inactivity
6-MWD† (m)	Normal (> 600)	< 600 - 200		< 200
BMI‡ (kg/m^2)	> 25	≤ 25 - 21		< 21

*Also severe if any of the following are present: repeated hospitalisation for exacerbations, co-morbidity, right heart failure, PaO_2 < 6.5 kPa, age > 65 years, respiratory acidosis.
†6-MWD = distance in metres walked in 6 minutes. Normal value in health > 600 m; moderate impairment < 300 m; severe impairment < 200 m. A change of 10% is considered clinically significant (see Annexure A).
‡BMI = body mass index, calculated as follows: mass in kg divided by height in m^2. A change in BMI of 1 kg/m^2 is considered significant.

in patients with COPD. Consequently, the FEV_1 expressed as a ratio of SVC (FEV_1/SVC%) improves the sensitivity of spirometry. Technical difficulties in assuring the quality and reproducibility of this manoeuvre limit its use. It is used by pulmonologists and those with suitable technical training.

6.1.1.2 Assessment of severity of airflow obstruction

To establish the severity of COPD, FEV_1 is expressed as a percentage of predicted values. The European Community for Steel and Coal (ECSC) predicted values (based on surveys performed in Europe) are recommended for routine use in South Africa.[9] It must however be noted that ethnic differences (amounting to approximately 12% lower values for FVC in Africans or African Americans) have been demonstrated in some series, and a correction factor (multiplication of the spirometric value by 0.9) might in some circumstances be advisable.

Measurement of peak expiratory flow (PEF), while helpful in suspected asthma, is not an appropriate test for diagnosing and evaluating the severity of COPD and does not distinguish obstructive from restrictive lung disease. It has limited use for patients who require monitoring to detect exacerbations, but is not used for assessing response to treatment.

6.1.1.3 Assessment of reversibility with short-acting bronchodilators

Spirometry should be performed before and 20 minutes after four puffs of a short-acting $beta_2$-agonist bronchodilator (e.g. salbutamol, fenoterol or terbutaline). An improvement in FEV_1 ≥ 12% from baseline and > 200 ml is considered to indicate a significant reversible component. Although reversibility of this magnitude is more common in asthma, it also occurs in a significant proportion of patients with COPD. However, in general, the larger the improvement the greater the likelihood that the diagnosis is asthma. Asthma is confirmed if the post-bronchodilator FEV_1 exceeds 80% of predicted.

A trial of systemic (oral) corticosteroids given for 14 days (Table V) forms part of the diagnostic assessment and is recommended in most symptomatic patients with severe airflow obstruction (FEV_1 < 50% of predicted) and particularly when asthma is suspected. A post-bronchodilator FEV_1 at the end of the steroid trial of > 80% of predicted confirms asthma. Partial improvement to less than 80% of predicted is more problematic to interpret, and suggests fixed airflow obstruction and a diagnosis of COPD. Partial improvement does not predict responsiveness to inhaled corticosteroids, but may be considered an indication for a strictly supervised trial of inhaled corticosteroids (see assessment of responsiveness to corticosteroids).

6.1.2 Assessment of severity of COPD

The assessment of severity of COPD is not based on spirometric measures alone, but on a combination of FEV_1 and several clinical indicators including severity of dyspnoea, functional impairment, 6-minute walking distance (6-MWD) and body mass index (BMI).[10] Grading of severity is used for prognostication and for selecting treatment. Details of the grades of severity are provided in Table IV.

6.1.3 Progression of disease and prognosis

The FEV_1 is a static measurement and only predicts prognosis when severely reduced (< 40% predicted).[6] Dyspnoea and

Table V. Trial of responsiveness to corticosteroids

- **Indications**
  1. Patients suspected to have COPD who have significant but partial reversibility with short-acting $beta_2$-agonists (to aid diagnosis).
  2. All patients with moderate or severe COPD (to aid selection of treatment)
- **Dose:** Prednisone 40 mg or methylprednisolone 30 mg once daily for 14 days, or inhaled corticosteroid equivalent to 800 µg budesonide daily for 6 weeks (400 µg twice daily)
- **Assessment of response before and after completion of treatment course**
  1. Detailed history of effort tolerance (with examples of activities)
  2. Measurement of FEV_1
  3. 6-MWD
- **Interpretation of results**
  1. Improvement of FEV_1 to ≥ 80% of predicted. Diagnosis is asthma . . . treat as asthma
  2. Partial improvement: Clear evidence of less dyspnoea/functional impairment and/or improvement of > 12% (and at least 200 ml) of baseline FEV_1, or an improvement of > 10% in the 6-MWD — add inhaled corticosteroids to treatment on a trial basis for a (further) 6-week period
  3. Little or no improvement (i.e. less than in 2): Reserve corticosteroids for acute exacerbations of COPD, but do not use as maintenance treatment
- **A short trial of corticosteroids will not reactivate tuberculosis.** If active tuberculosis is suspected and in cases where long-term steroid therapy is planned, an initial chest radiograph is advised

functional impairment are linked because grade of dyspnoea is allocated according to the activities that provoke it, and dyspnoea is the symptom that most commonly limits activities.[11] Together, dyspnoea and functional impairment predict prognosis. The 6-MWD is another more objective and reproducible measure of functional impairment and can be used to follow the progression of disease. In the absence of other cause of weight loss, a decline in BMI is associated with advanced disease. A value of < 21 kg/m^2 is associated with a 50% 5-year mortality.[10]

FEV_1 declines slowly at a rate of 50 - 60 ml per year.[12] Rapid symptomatic deterioration may result from the development of complications, such as pulmonary thrombo-embolism, right heart failure and ischaemic heart disease with left ventricular failure.

6.1.4 Chest radiography

The chest radiograph on its own is not diagnostic, and has a high false-positive rate. Moreover, a normal chest radiograph does not exclude the diagnosis of COPD. A chest radiograph is therefore not essential for the diagnosis unless additional pathologies are suspected. The presence on chest radiograph of structural disease such as bullae and scarring from previous tuberculosis increases the likelihood that airflow limitation, if present, will be irreversible.

6.1.5 An integrated management plan for COPD

An integrated management plan for the management of COPD, based on the grades of severity, is provided in Table VI.

- **Stage 0 — normal, but at risk ($FEV_1 \geq 80\%$ of predicted).** Patients are asymptomatic, but are exposed. The strategy is to prevent further damage. Smoking and other risk factors must be stopped and avoided.
- **Stage 1 — mild COPD (FEV_1 60 - 79% of predicted).** Patients may be asymptomatic; treatment is prevention of further exposure (as above), and of progression factors, e.g. exacerbations. Bronchodilators: short-acting inhaled $beta_2$-agonist or anticholinergic, or a combination inhaler taken as needed according to symptoms. Oral theophylline can be added if symptoms are more regular or persistent.
- **Stage 2 — moderate COPD (FEV_1 40 - 59% of predicted).** Symptoms are more persistent and limit activities; treatment

Table VI. Integrated management plan for COPD

Stage of COPD	Prevention	Bronchodilators	Other drugs	Other measures
Stage 0 $FEV_1 \geq 80\%$	Education Avoidance measures: smoking cessation			
Stage 1 FEV_1 60 - 79%	As above	On demand inhaled short-acting $beta_2$-agonist or anticholinergic broncho-dilator alone or combination inhaler or oral theophylline		
Stage 2 FEV_1 40 - 59%	As above	Regular use of inhaled short-or long-acting bronchodilator, alone or in combination +/- oral theophylline. Two or 3 might be needed with increasing severity. Long-acting $beta_2$-agonists include formoterol and salmeterol, and long-acting anticholinergic, tiotropium	A trial of oral or inhaled corticosteroid may be considered where FEV_1 is below 50% of predicted and if objective benefit (in FEV_1 and effort tolerance) is found, or the patient has frequent exacerbations of COPD (3 or more per year) (See notes on corticosteroids)	Prevention of exacerbations: • Influenza vac-cination annually • Regular broncho-dilators (with inhaled corticosteroids in a minority) Rehabilitation: • Education • Exercise programme • Psychological support • Nutrition
Stage 3 $FEV_1 < 40\%$	As above	As above	As above	• Domiciliary oxygen • Treatment of cor pulmonale • Treat complications and co-morbidity

as above, plus one or more bronchodilators taken regularly. Theophylline may be added. A long-acting bronchodilator may be considered (beta$_2$-agonist or anticholinergic). Inhaled corticosteroids, alone or in combination, should be used only in selected cases. Rehabilitation is important.

- **Stage 3 — severe COPD (FEV$_1$ < 40% of predicted).** Symptoms are persistent and limit even the lightest daily activities such as bathing, dressing and walking a few metres; treatment as for stage 2. Use of a combination of bronchodilators, taken regularly, is advised. A trial of corticosteroids is recommended and either oral or inhaled maintenance treatment may be considered. Prevention of exacerbations is essential. Treatment of chronic respiratory failure (hypoxaemia) with domiciliary oxygen, cor pulmonale and other complications may be required. Rehabilitation is essential.

6.2 Smoking cessation (primary and secondary prevention)

Smoking cessation is the only measure that has been shown to slow progression of COPD, and is one of the most cost-effective interventions in health care. All smokers, and particularly those with COPD, must be encouraged to stop smoking, and all health care workers should be familiar with and promote smoking cessation among their patients.

Avoidance of occupational and atmospheric pollution, including passive or 'side-stream' tobacco smoke exposure is desirable for all, but particularly important for susceptible persons, namely pregnant women, infants and children, and persons with COPD, especially those with alpha-1-anti-protease deficiency.

The addictive nature of nicotine is attributable to its unique physiological effects on the central nervous system, i.e. both stimulation and relaxation and improved concentration and vigilance. Appetite suppression aiding weight control is an additional attraction for many. Together, these effects persuade many to continue smoking in the face of compelling evidence of its harmful effects.

Benefits of smoking cessation in patients with COPD. Although much lung damage caused by smoking is irreversible, all patients must be encouraged to stop smoking, regardless of the severity of their disease. Advantages of cessation include:

1. Slowing of the rate of decline of lung function to that of normal ageing. A small improvement in spirometry occurs in some.

2. Improved oxygen transport in blood, through reductions in carboxyhaemoglobin, and blood viscosity.

3. Reduced tendency to thrombosis.

4. Improvements in appetite, body mass, muscle strength and exercise tolerance.

5. Possible improved efficacy of some classes of drugs used in COPD.

6. Delayed development of respiratory failure and cor pulmonale.

6.2.1 Smoking cessation programmes

The plethora of smoking cessation methods advertised in the popular and medical press reflects the poor efficacy of most in overcoming the addictive effects of nicotine. Many are behavioural in approach, but some are pharmacological, and involve gradual weaning from nicotine through nicotine replacement, with or without mood modifiers to counteract the negative effects of withdrawal. Many interventions show impressive short-term results, but long-term abstinence (defined as cessation without relapse that lasts for 12 months) occurs in fewer than 30% of patients (for most methods only 15 - 20%). Factors associated with poor success include multiple previous attempts, heavy smoking and relapse within the first 2 weeks.

6.2.2 Features that contribute to a successful programme

1. An initial in-depth interview to discuss the patient's smoking habits and previous quit attempts and that provides information and advice on cigarette brands (including tar and nicotine content), withdrawal symptoms and coping strategies. Cessation advised by a doctor, particularly if repeated on several occasions, has been shown to be more effective than advice offered by other health care professionals. Several medical aids reimburse smoking cessation counselling.

2. Abrupt cessation rather than gradual smoking reduction, with agreement on a 'quit date' and provision of support for adherence to the commitment (by phone call).

3. Reinforcement and follow-up. Some programmes recommend a schedule of follow-up visits, e.g in weeks 1, 2, 4, 8, 12, 16, 20, 26 and 52, but support provided during usual visits to clinic or rooms is also effective.

4. Nicotine replacement is advised for patients who are unsure of their ability to stop, where there are signs of severe addiction, and/or if severe withdrawal symptoms have been experienced during a previous attempt. Options are a sublingual spray, inhaler, gum and patches. Clinicians should be familiar with the advantages and disadvantages of each method, potential medical complications with their use, recommended dosing schedules, and limits to their use.[13]

5. Additional pharmacological support: use of bupropion improves the quit rate. Nortriptyline is of limited benefit. Buspirone and clonidine are ineffective.

Clinical trials examining the efficacy of hypnosis and acupuncture have shown no additional benefit of these treatments.

6.2.3 Awareness programmes

Awareness programmes serve to protect the public from cigarette smoking and other harmful exposures, with the particular aim of protecting susceptible individuals (children, asthmatics and individuals with impaired lung function).

6.2.4 Other preventive measures

These include practical measures to avoid exposure to cold and influenza at home and work and include annual influenza and 5-yearly pneumococcal vaccination.

6.3 Improvement of breathlessness (treatment of airflow obstruction)

6.3.1 Mild disease

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

- **Beta$_2$-agonist inhaled** (e.g. salbutamol, fenoterol and terbutaline, approximately 6-hourly as needed, administered via metered dose inhaler (MDI) (dose 2 puffs) or dry powder inhalation device (DPI) (single dose).

  OR
- **Anticholinergic inhaled** (e.g. ipratropium — administration and dose as above).

  OR
- **Combination MDI containing short-acting anticholinergic** (e.g. ipratropium and beta$_2$-agonist — administration and dose as above).

  OR
- **Regular oral slow-release theophylline** (200 - 400 mg twice daily or 400 - 800 mg at night as a daily dose).

6.3.2 Moderate and severe disease

First consider a trial of oral or inhaled corticosteroids (see below). This forms part of the diagnostic assessment and is an indicator of likely responsiveness to therapy. It also establishes a target lung function for bronchodilator treatment and should be performed unless a medical contraindication exists. Patients should be in a stable phase.

Include combinations of bronchodilators (usually at least 2 with different modes of action and combined effect). One or more of the following combinations should be considered:

- **Regular beta$_2$-agonist and anticholinergic** either from a single combination MDI or from separate MDI (or DPI) devices: usually 2 puffs (up to 4 in severe cases) approximately 6-hourly as needed.

  OR
- **Regular long-acting beta$_2$-agonist** (e.g. salmeterol or formoterol 12-hourly) with short-acting inhaled bronchodilator 'rescue' as needed.

  OR
- **Regular long-acting inhaled anticholinergic** (e.g. tiotropium) with short-acting inhaled beta$_2$-agonist 'rescue' as needed.
- Any of the above may be combined with **regular oral slow-release theophylline** given either once or twice daily (200 - 400 mg twice daily or 400 - 800 mg daily, take at night).

6.3.3 Notes on bronchodilators

6.3.3.1 General principles

- Most patients with COPD have a degree of reversible airways obstruction and display hyperresponsiveness on bronchial challenge.
- Most patients respond with partial improvement in FEV_1. In some there are also improvements in FVC, inspiratory capacity and SVC and reduction in gas trapping.
- In many patients there is a partial relief of symptoms, improved quality of life and a reduction in the frequency and/or severity of exacerbations. The magnitude of these benefits is usually less than in asthma.
- Bronchodilators do not alter the progressive decline in FEV_1.
- Even patients who do not demonstrate a bronchodilator response on spirometric testing should be given a trial of bronchodilator treatment as breathlessness may be improved by a number of mechanisms.
- The main classes of bronchodilators are beta$_2$-agonists, anticholinergics and theophylline.
- The development of long-acting bronchodilators (beta$_2$-agonists and anticholinergics) represents a significant advance in the treatment of COPD.
- Bronchodilators may be most effective when used in combinations that exploit their different mechanisms of action.
- A measured FEV_1 response to a single dose of bronchodilator does not predict long-term response.
- In all but mild COPD their use must be regular and long-term rather than 'as needed' as in asthma.
- The addition of more bronchodilators to the treatment regimen in individual patients is 'symptom-driven', that is, bronchodilators are added until maximal relief of breathlessness and/or improvement in effort tolerance and quality of life is achieved or dose is limited by side-effects.
- Because individual patients respond differently to the various classes of bronchodilators, and responses cannot be

reliably predicted, a process of trial and review is recommended to establish which drugs in combination provide the best result in each patient. This usually takes several visits over months, but ensures that the most cost-effective drugs and combinations are used.

6.3.3.2 Delivery devices for bronchodilators

- Inhaled bronchodilators are preferred over oral agents as they:
  - are generally more effective
  - are more conveniently titrated
  - have fewer side-effects.
- **The pressurised metered dose inhaler** (pMDI) operated by the patient using the 'push-and-breathe' method is the most widely accepted and used method of delivery of inhaled bronchodilators (and corticosteroids) and at lowest cost.
- Up to 40% of patients, and possibly more in the elderly and those with severe disease or physical limitations such as arthritis, have difficulty using these devices, particularly co-ordinating actuation with inspiration. Consequently they derive little or even no benefit in spite of adherence to dosing schedules. Since this is also the group of patients in whom the greater systemic effects of oral bronchodilator use are most significant (tachycardia, arrhythmias, tremor and gastro-intestinal effects), other methods of delivering inhaled drugs should be considered.
- **Spacer devices.** These are widely used for delivery of inhaled corticosteroids because they reduce oral deposition of drug. However, their size makes them unsuitable for bronchodilators, which in general need to be carried about during the day for frequent use, and mastery of the technique of use is a problem for some.
- **Breath-activated pMDIs and powder devices** overcome the above problems but are more costly.
- **Nebulisers** are an option for delivering high doses of bronchodilator in patients with advanced disease, poor inhalation technique and/or during exacerbations. Nebulised ipratropium plus $beta_2$-agonists can be used 3 or more times daily. In general, nebulisers tend to be overused in COPD, resulting in unnecessary expense. Patients requiring nebuliser therapy should, where possible, be assessed by a specialist for review of their treatment.
- It is vital to ensure that the individual is able to use the delivery device that is prescribed. This should be checked at regular intervals during follow-up visits.

6.3.3.3 Anticholinergics

- Bronchodilatation achieved with anticholinergics results from inhibition of muscarinic receptors in the lung, thereby reducing airway tone, and relieving bronchospasm. The location and distribution of muscarinic receptors in the lung differs from that of $beta_2$-receptors, and their functional role may be more important in the elderly and in smokers. This might account for their apparent superior efficacy over other classes in the elderly and smokers, observed in some studies.
- They have a slower onset of action than rapid-acting $beta_2$-agonists (± 40 minutes to peak effects versus 10 - 20 minutes) but are effective for longer (6 hours for ipratropium bromide, and more than 24 hours for tiotropium).
- Efficacy is dose-dependent, and the dose of ipratropium can be safely increased to obtain a better effect.
- Their use is associated with fewer side-effects, especially in COPD and the elderly, and unlike $beta_2$-agonists which may be associated with tachycardia, palpitations, tendency to cause hypoxaemia and tachyphylaxis. A small proportion of patients experience dryness of the mouth. No impairment of mucociliary clearance has been reported. Significant urinary or pupillary effects are uncommon, even in high doses and in the elderly.
- They may be safely combined with $beta_2$-agonists.
- **Long-acting anticholinergic (tiotropium).** Tiotropium is an anticholinergic that causes prolonged and selective blockade of human M1 and M3 receptors. Its clinical effect in patients with moderate and severe disease is superior to ipratropium without increasing side-effects. Improvement in lung function is greater and sustained over 24 hours, permitting once-daily dosing. It is at least as effective as long-acting $beta_2$-agonists and is superior for certain end-points, including duration of action. It is available only in powder form via a Handihaler device.

6.3.3.4 $Beta_2$-agonists

- **Short-acting $beta_2$-agonists.** These have a rapid onset of action and achieve similar effects to anticholinergics. They may be used regularly and as monotherapy. Examples include salbutamol, fenoterol and terbutaline.
- **Long-acting $beta_2$-agonists.** The group includes salmeterol (given in a dose of 50 µg twice daily) and formoterol (in a dose of 9 or 18 µg twice day). Regular use of doses higher than these is not recommended. Combining long-acting $beta_2$-agonists with inhaled steroids may provide additional benefits in terms of symptom control and reduction in exacerbations. However this combination should not be considered first-line therapy.

6.3.3.5 Oral theophylline

- Theophylline has less of a bronchodilator effect than anticholinergics and $beta_2$-agonists but has several additional therapeutic benefits including a measurable

effect on markers of airway inflammation in COPD. Other benefits are low cost, and oral route of administration, which is preferred by some patients and may improve compliance. Disadvantages are toxicity (particularly in the elderly), drug interactions and variable metabolism.

- They are better tolerated if started at a low dose (half or one tablet daily for a week) and then increased to the full therapeutic dose, and given after a meal. Initial gastro-intestinal side-effects like anorexia, nausea and change in bowel habit may improve with continued use.
- Sustained-release tablets (both twice- or once-daily formulations are available) are recommended for maintenance treatment.
- Theophylline can be safely used in combination with other classes of bronchodilator.
- 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 to certain categories of patients is as follows: (*i*) smokers and patients on phenytoin therapy — increase dose by one-third; and (*ii*) patients with congestive cardiac failure or liver disease, the elderly, and those on most macrolide antibiotics, ciprofloxacin or cimetidine — decrease the dose by one-third.
- Combination tablets containing theophylline and other bronchodilators or sedatives are not recommended.

6.3.3.6 Comparison of different combinations of bronchodilators

A step-wise increase in inhaled and oral agents with the final combination determined by the maximum functional response achieved in each individual should be followed. This may include combinations of short-acting beta$_2$-agonist and short-acting anticholinergic, long-acting beta$_2$-agonist with short-acting anticholinergic, or long-acting anticholinergic with a short-acting beta$_2$-agonist. The potential benefit of combining a long-acting beta$_2$-agonist with tiotropium has not been studied, and is an expensive option. Although both the long-acting beta$_2$-agonists and anticholinergics are more expensive than their short-acting counterparts they achieve and maintain a higher level of clinical benefit (less dyspnoea, improved effort tolerance, improved FEV_1 and fewer exacerbations). Owing to their cost they should not be used as first-line therapy. They are recommended for patients with severe or symptomatic moderate grades of COPD. Consideration should be given to stopping them if no symptomatic benefit is evident. Oral theophylline may be added to any combination of inhaled treatment. The effects of these and other drug combinations on survival in patients with severe COPD has not been confirmed, but long-term studies are underway.

6.3.4 Notes on corticosteroids

6.3.4.1 Trial of corticosteroids: Assessment of responsiveness to corticosteroids (systemic or inhaled)

A trial of oral corticosteroids is indicated in patients with significant persistent breathlessness as an aid to distinguishing COPD from asthma and deciding on the need for continued treatment with inhaled or even oral corticosteroids. A 6-week trial of high-dose inhaled corticosteroids is an alternative for this purpose (Table VI). This must be monitored with spirometry. Another objective test of response is the Six Minute Walk Distance (6-MWD) (see Annexure A).

6.3.4.2 Chronic oral corticosteroids

Chronic oral corticosteroid use improves symptoms and lung function in a small proportion of patients, but because of side-effects should be used with caution, and only when there is objective evidence of improvement in patients tested during a period of relative stability. When initiated, regular attempts should be made to reduce treatment to the lowest dose that is not associated with acute exacerbations or worsening breathlessness, and a dose of greater than 7.5 mg of prednisone (or equivalent) is seldom beneficial or indicated. Sustained use, and repeated short high-dose courses are associated with severe side-effects, particularly in the elderly. The development of muscle weakness and osteoporosis with vertebral collapse is common. Both further limit activity and accelerate functional decline. Where continued use is indicated, a switch to inhaled corticosteroids (equivalent to 400 mg budesonide twice daily) should be attempted, starting with a 6-week trial period (see above).

6.3.4.3 Inhaled corticosteroids

The use of inhaled corticosteroids in COPD remains controversial. Although there is evidence of airway inflammation in COPD, the benefits of long-term preventive inhaled corticosteroid treatment, as recommended for asthma, are limited. In steroid-withdrawal trials and in prospective trials involving their addition to bronchodilator therapy, their use has been associated with the reduction of frequency and/or severity of acute exacerbations of COPD, and symptomatic improvements in a minority of subjects. Initial results of trials in combination with long-acting beta$_2$-agonists suggest some additive benefit.

6.3.5 Mucolytics and mucokinetic agents

Expectoration of tenacious sputum is a distressing symptom. Mucolytics, mucokinetic drugs and cough syrups (oral and inhaled) have not been shown to be effective, and are not recommended. Regular dosing with oral acetylcysteine has provided limited benefit in some but not all studies.

6.3.6 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 does not form part of routine management in stable patients.

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

6.3.7 Other anti-asthma drugs

Other anti-asthma drugs, e.g. sodium cromoglycate, ketotifen and nedocromil sodium, are of no value in COPD.

6.3.8 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.

6.3.9 Other procedures and techniques

'Therapeutic' bronchoscopy and pulmonary lavage have no place in the routine management of COPD.

6.4 Improving quality of life (pulmonary rehabilitation and education)

Improvement in health status in COPD is recognised as one of the most important goals of therapy, since it reflects the patient's perspective and may be more clinically relevant than a physiological endpoint like FEV_1. Health status questionnaires are now routinely used in studies of drugs and other treatment modalities. Several drug combinations recommended above are also associated with sustained benefit to quality of life and may slow the rate of deterioration. Long-term domiciliary oxygen therapy also improves health status and if used appropriately on selected patients, prolongs life.

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

6.4.1 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
- A 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.

6.4.2 Physical conditioning

All patients, regardless of age, who are adequately motivated, should be included in rehabilitation programmes. In its simplest form physical conditioning should include a graded programme of upper and lower limb exercise, e.g. free-range walking. Cycling and treadmill walking are useful alternatives. Hypoxic patients should be exercised with caution. Supervised programmes designed to improve both muscle endurance and strength, particularly in the muscles of the extremities and thorax, result in improvements in the 6-MWT, reduced dyspnoea and improved quality of life, and recovery after exacerbations may be more rapid.

Initial exercise programmes are individualised according to the patient's disease severity and mobility. In the first weeks the duration and intensity of exercises are increased to a level that is sustainable for a total of 15 - 45 minutes per session. Four or more sessions per week are recommended, at least some of them under supervision. Such programmes last 6 - 8 weeks, but patients must be encouraged to continue the exercise prescription indefinitely, or the benefit will be temporary.

For more severe disease, breathing exercises, e.g. pursed-lip breathing to improve airflow during expiration, '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.

6.4.3 Psychological support

This involves group and family support and advice to the family from the practitioner and other caregivers.

6.4.4 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. Anabolic steroids improve body mass in patients with weight loss, but do not have a sustained beneficial effect on respiratory muscle strength or effort tolerance.

6.4.5 Long-term domiciliary oxygen therapy (LTDOT)

The purpose of oxygen therapy is to correct hypoxaemia, thereby preventing the effects of the latter on various organ systems. The full benefit is therefore only evident in patients with persistent hypoxaemia. In such patients it has been shown to reduce the complications of respiratory and right heart failure and to improve survival.

6.4.5.1 Indications for LTDOT

- Non-smokers with stable, severe COPD (usually but not exclusively with an FEV_1 of less than 1.5 l and $FEV_1/FVC < 70\%$)
- Arterial hypoxaemia (PaO_2 less than 7.3 kPa or 55 mmHg) or oxygen saturation < 90% at rest
- With or without hypercapnia ($PaCO_2$ greater than 6.0 kPa or 45 mmHg)
- With or without oedema.

Severe airflow obstruction (low FEV_1 and FEV_1/FVC) must be confirmed since LTDOT is not of proven benefit in other forms of disease. Before prescribing LTDOT, the patient's COPD must be in a stable phase on optimal drug treatment. Hypoxaemia must be confirmed by arterial blood gases performed while the patient is breathing room air. Following exacerbations of COPD, spirometry and PaO_2 may continue to improve for up to 3 months. Therefore in all patients these should be checked 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. Hypoxaemia is assessed with the patient at rest. Patients in whom a fall in PaO_2 occurs only during exercise and sleep do not benefit from LTDOT.

Patients and their families need to understand both the purpose of the treatment, and the need to use it for at least 16 hours per day. Other components of rehabilitation must be provided.

Worsening hypercapnia caused by hypoventilation is an occasional complication of oxygen therapy in patients with severe COPD and hypercapnic respiratory failure. Oxygen administration in such patients must be carefully controlled (initial flow rates through nasal cannulae, or facemask concentration of oxygen should be kept low until the effect on the arterial $PaCO_2$ confirms the absence of deterioration).

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.

6.4.5.2 Oxygen prescription

Oxygen is administered by facemask or nasal cannula for a total of at least 16 hours per 24-hour day. A flow rate of 1 - 2 l/minute is used, the rate being determined in each case by arterial blood gas determinations. Oximetry may be used for follow-up checks.

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

6.4.5.3 Palliative symptomatic oxygen therapy

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

6.5 Prevention and treatment of exacerbations of COPD

6.5.1 Definition, natural history and diagnosis of exacerbations

An exacerbation is defined as an increase in symptoms and signs of COPD above the usual day-to-day variation expected by the patient. Exacerbations vary both in severity and frequency. Most patients experience 1 or 2 episodes per year, but some have many. They tend to become more common as COPD progresses. Exacerbations are important clinical events in COPD, severely affect quality of life, are associated with permanent worsening of COPD in some patients, and account for a large percentage of the direct costs associated with the treatment of COPD. Their prevention is an important goal of treatment.

The main symptom of an exacerbation is increased breathlessness, often accompanied by wheezing, chest tightness and increased cough and sputum. They are often associated with signs of airway infection, increased volume and colour change in sputum (to yellow or green), and fever. Other symptoms are malaise, drowsiness, insomnia, fatigue, depression, confusion and fever.

The most common causes of exacerbations include:

- tracheobronchial infections
- environmental (atmospheric) pollution (including cigarette smoke and allergen load)
- weather changes
- aspiration associated with gastro-oesophageal reflux.

The role of bacterial infection and the place of antibiotics in the treatment of exacerbations is controversial. Potentially pathogenic bacteria are found in about 50% of exacerbations, and viruses in a further proportion of episodes. In most cases they are likely to be relevant, and reports confirm the benefit of antibiotic treatment on the rate of recovery, morbidity, and even hospital stay, particularly in patients with severe COPD, and those who have severe exacerbations.

Acute exacerbations must be distinguished from other diseases and complications of COPD, including pneumonia, pneumothorax, congestive heart failure, arrhythmia and pulmonary embolism that require alternative treatment. When exacerbations are associated with features of infection (pyrexia and purulent sputum), the presence of pneumonia and other forms of lower respiratory infection must be excluded using chest X-ray. An approach to the treatment of common tracheobronchial infections is provided in Annexure B.

6.5.2 Prevention of exacerbations

The following measures have each been shown to reduce the frequency and/or severity of COPD exacerbations:

- Smoking cessation
- Prevention of respiratory infections (influenza and pneumococcal vaccination)
- Bronchodilators: regular dosing with ipratropium bromide, theophylline, long-acting beta$_2$-agonist, tiotropium, and in various combinations described above
- Oral and high-dose inhaled corticosteroids alone, and in combination with bronchodilators, particularly the long-acting varieties.

6.5.3 Management of exacerbations

6.5.3.1 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 impulse oximetry (if blood gas is not available), chest radiograph, ECG and theophylline level (if the patient has been on theophylline).

6.5.3.2 Treatment

Bronchodilators

- First-line treatment is nebulisation with the combination of ipratropium bromide and beta$_2$-agonist (salbutamol or fenoterol) given 4-hourly (or as often as every 30 - 60 minutes or continuously in severe cases). Early conversion to an MDI is desirable. Alternatively, multiple actuations of an MDI delivered via a spacer device may be as effective as nebulisation.
- Theophylline. Should there be inadequate sustained response to the above treatment, intravenous aminophylline may be used.

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 clear evidence or strong suspicion of infection (marked sputum purulence and/or fever), and in those with severe COPD or a severe exacerbation. In such cases, sputum Gram stain may be of help by confirming the presence of relevant organisms, but is not an essential investigation. The organisms most commonly involved are *Streptococcus pneumoniae*, *Haemophilus influenzae* and *Moraxella catarrhalis*. With the increasing appearance of resistance to penicillin and macrolides of the pneumococcus, beta-lactam resistance of *H. influenzae* and *M. catarrhalis*, local sensitivity data should be considered when chosing an antibiotic. In areas where there are high levels of resistance to the macrolides and doxycycline, these agents should be avoided. Alternatives such as amoxycillin/clavulanate, cefuroxime, or quinolones may be used. Intravenous administration is preferred for severe illness and in the case of pneumonia on chest radiograph. Duration of treatment should be 10 - 14 days for severely ill patients or for persistent infections.

Oxygen therapy

Oxygen should be started at 24% or 1 - 2 litres/minute by nasal cannula. 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: See treatment of right heart failure.

Heparin prophylaxis: See anticoagulant treatment.

Physiotherapy to assist with clearance of secretions. Postural drainage and chest percussion are of limited benefit except in patients with bronchiectasis and may cause distress. Treatment is most useful after nebulisation with a bronchodilator.

6.5.3.3 Hospital admission

Indications for hospital admission in patients with COPD:

1. **An acute exacerbation** associated with one or more of the following features:

- 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 and new or worsening hypercapnia
- Newly occurring arrhythmia
- Elderly or frail patients.

2. New or worsening **right-sided cardiac failure** unresponsive to outpatient management.

Indications for ICU admission:

The usual indication is respiratory failure. However, it is frequently necessary to admit patients with other diseases because they have COPD.

Pre-conditions for ICU admission:

1. Satisfactory functional status before the exacerbation (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 mmHg) on room air
- Arterial blood pH < 7.3
- Confusion.

3. The presence of a reversible factor. Examples are infections, bronchospasm, oxygen-induced carbon dioxide narcosis, sedative administration or other associated illnesses.

Ventilatory support must be considered for patients with one or more of the following features:

- Hypoxaemia (PaO_2 < 50mmHg) despite supplemental oxygen
- Exhaustion, confusion, coma
- pH < 7.3 and declining (respiratory acidosis)
- Respiratory or cardiac arrest
- Inability to clear secretions.

Note: Patients with severe COPD may have chronic severe dyspnoea, hypercapnia and hypoxaemia that is stable and cannot be improved. Therefore the presence of these features must not be viewed in isolation, as an indication for ventilatory support. Rather, ventilation is indicated for acute deteriorations from baseline. Prior knowledge of a patient's blood gases and clinical status in the stable state is therefore a valuable aid.

Modalities of ventilatory support include invasive (mechanical ventilation) and non-invasive continuous positive airways pressure (CPAP) or BiPAP techniques.

Before discharge, the following require attention and must be provided:

- Education on COPD (see above)
- Further need for oxygen (short-term or long-term domiciliary oxygen — see above)
- A written home management action plan
- Outpatient follow-up appointment
- Rehabilitation plan
- A plan for smoking cessation
- Assessment of home conditions and psychosocial support.

6.6. Prevention and treatment of complications

The main complications that need to be treated include right heart failure, severe exacerbations, pulmonary embolism, pneumothoraces and chronic respiratory failure not related to exacerbations.

6.6.1 Treatment of right heart failure

The management of right heart failure may be summarised as follows:

- Identify and treat the cause. In COPD this might include an acute respiratory infection, worsening airflow obstruction (review bronchodilator and other treatment) or worsening hypoxaemia from additional factors such as a move to higher altitude or a thrombo-embolic event.
- Administer oxygen if hypoxaemic.
- Diuretics (e.g hydrochlorothiazide 25 mg or equivalent). Avoid large decreases in preload which may precipitate hypotension.
- Cardiac glycosides must be avoided except in the presence of atrial fibrillation and/or left ventricular dysfunction/failure.
- ACE inhibitors and calcium antagonists are not indicated in the management of cor pulmonale or right ventricular failure.

6.6.2 Anticoagulant treatment

6.6.2.1 Heparin prophylaxis

Prophylactic subcutaneous heparin to prevent deep-vein thrombosis in indicated during acute exacerbations where patients are immobile for prolonged periods.

6.6.2.2 Long-term anticoagulant treatment

Chronic treatment with warfarin needs to be considered in patients with pulmonary hypertension and right heart failure, in those with atrial fibrillation, and in patients with a high haematocrit, particularly if it recurs after venesection. It should also be considered in patients with unexplained hypoxic episodes where pulmonary thrombosis or thrombo-embolism is suspected. Confirmation of this diagnosis is seldom possible, but spiral CT scans may occasionally be diagnostic.

6.6.3 Miscellaneous

Pneumothorax: The development of a spontaneous pneumothorax must be considered when patients with stable COPD suddenly deteriorate, as patients with respiratory impairment tolerate even a small pneumothorax poorly.

7. Additional considerations

7.1 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 erythrocytes are found.

Referral for full sleep study (polysomnography) and specialised opinion should only be considered if sleep-disordered breathing is suspected (i.e. daytime hypersomnolence and other symptoms of sleep deprivation, or a strong history of loud snoring with apnoeic events).

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

7.2 Surgery for emphysema

Giant bullectomy is occasionally indicated.

Lung volume reduction surgery (LVRS: bilateral non-anatomical excision of emphysematous lung tissue) is a useful modality of therapy in a small, highly selected group of patients. These patients must be on optimal medical therapy, severely symptomatic with dyspnoea, and have evidence of marked air trapping and predominantly upper lobe emphysema (confirmed by high-resolution CT scan). Careful selection and preparation for the procedure is critical. If this is inadequate, mortality is high and results are poor. Potential patients must be referred to a pulmonologist participating in a multidisciplinary LVRS programme for evaluation. All patients must undergo a 6-week exercise conditioning programme before surgery. It must be explained that the procedure is not curative, and that any benefit is temporary and will last for only a few years (usually 2 or 3).

7.3 COPD and surgery

Patients with COPD are at high 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. An assessment of exercise tolerance, either as a formal exercise test or through scrutiny of ability to climb several flights of stairs, is a more reliable predictor of surgical risk than FEV_1, for both abdominal and thoracic surgery. All patients with persistent dyspnoea, (stages 2 and 3) require these investigations before surgery.

7.4 Air travel

The risks of air travel for patients with COPD include:

- Worsening hypoxaemia at altitude. Most commercial aircraft are pressurised to an altitude of between 2 250 and 3 250 m. This altitude may have little effect on patients who live at altitudes of 2 000 m (e.g. on the Highveld), but for those who are hypoxaemic at sea level, this may result in severely symptomatic hypoxaemia. Arterial PO_2 decreases by 1 - 1.5 kPa for every 1 000 m altitude.
- Increased physical stress of travel (airport transfers, luggage, etc).
- Risk of contracting a respiratory infection.
- Increased altitude at destination causing increased dyspnoea.

Patients need to balance the potential discomforts and risks against the purpose 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 who 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.

8. Process

In July 2001, the Council of the South African Thoracic Society resolved to revise the 1996 COPD Guideline and appointed a convenor and editorial committee, selected a review date and agreed on the format of the process of revision. The working group comprised most members of the previous working group. The editorial committee identified areas of the guideline requiring revision and commissioned members of the working group to prepare reviews of new data in these areas, with particular reference to the content of the 2001 version of the Global Obstructive Lung Disease Guideline. The workshop was held at Sun City, North West province, in August 2002. A

draft revision document based on the consensus obtained at the workshop was compiled by the editorial board and circulated for further comment from the guideline committee, in May 2003. These further comments were included (as considered appropriate by the editorial committee), and the final draft was then considered and approved by the Council of the South African Thoracic Society by postal vote in June 2003.

9. References

1. O'Brien J, Feldman C, Bateman ED, Plit M. Guidelines for the management of chronic obstructive pulmonary disease. For a Working Group of the South African Pulmonology Society. *S Afr Med J* 1998; **88:** 999-1010.
2. Global Obstructive Lung Disease initiative (GOLD). *Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Pulmonary Disease*. NHI publication no.2701, Bethesda Md: National Institutes of Health, 2001.
3. *South African Demographic and Health Survey 1998: Full Report*. Medical Research Council of South Africa, Department of Health, 2002.
4. Hnizdo E, Singh T, Churchyard G. Chronic pulmonary function impairment caused by initial and recurrent pulmonary tuberculosis following treatment. *Thorax* 2000; **55:** 32-38.
5. Lundback B, Lindberg A, Lindstrom M, *et al*. Not 15 but 50% of smokers develop COPD? - Report from the Obstructive Lung Disease in Northern Sweden Studies. *Respir Med* 2003; **97:** 115-122.
6. Anthonissen NR. Prognosis in chronic obstructive pulmonary disease. Results from multi-center clinical trials. *Am Rev Respir Dis* 1989; **140:** 595-599.
7. Roberts CM, Lowe D, Bucknall CE, *et al*. Clinical audit indicators of outcome following admission to hospital with acute exacerbation of chronic obstructive pulmonary disease. *Thorax* 2002; **57:** 137-141.
8. Van Schalkwyk EM, Schultz C, Joubert JR, White NW. Guide for office spirometry in adults. *S Afr Med J* 2004; **94:**
9. Quanjar PH, Tammeling GJ, Cotes JE, *et al*. Lung volumes and forced ventilatory flows: report of the working party standardization of lung function tests. European Respiratory Society. *Eur Respir J* 1993; **6:** suppl. 16, 5 - 40.
10. Schols AM, Slangen J, Volovics L, Wouters EFM. Weight loss is a reversible factor in the prognosis of chronic obstructive pulmonary disease. *Am J Respir Crit Care Med* 1998; **157:** 1791-1797.
11. Nishimura K, Izumi T, Tsukino M, Oga T. Dyspnea is a better predictor of 5-year survival than airflow obstruction in patients with COPD. *Chest* 2002; **121:** 1434-1440.
12. Anthonisen NR, Connett, Murray RP. Smoking and lung function of lung health participants after 11 years. *Am J Respir Crit Care Med* 2002; **166:** 675-679.
13. Raw M, McNeill A, West R. Smoking cessation guidelines for health professionals. *Thorax* 1998: **53:** (suppl. 5), S1-S19.

Annexure A. The Six-Minute Walk Distance (6-MWD)

The 6-MWD provides an objective reproducible assessment of the combined function of lung, heart and proximal muscle endurance. The unit of measurement is total distance covered during 6 minutes (in metres). Level of dyspnoea during the test may be assessed using the Borg scale (Table VII). A major use of the test is for following the course of patients over time and evaluating the effects of changes in treatment.

Table VII. Borg scale for assessing of breathlessness

0	No breathlessness
1	Very slight
2	Slight
3	Moderate
4	Somewhat severe
5	Severe
6	
7	Very severe
8	

Procedure

A 20 - 30 m indoor course is marked out with distance markers. A treadmill may be used. The same method must be used for subsequent follow-up tests.

Patients must wear comfortable clothing and shoes, and take their usual medication on the test day. Those on long-term oxygen may perform the test with oxygen, provided that a suitable long supply line or mobile oxygen source is provided and carried by an assistant (not the subject). As there is a learning effect, the first test performed by each patient should be disregarded, and a second, performed on a different day, regarded as the baseline value.

On arrival **on the test day**, the subject must rest in a chair for 10 minutes. Resting dyspnoea level, pulse, blood pressure and oxygen saturation must be recorded.

Safety issues: Testing must be deferred in patients with a resting pulse rate > 140 beats per minute, diastolic blood pressure readings of > 110 mmHg, unstable angina, or myocardial infarction during the previous month. Monitoring of pulse rate by means of pulse rate monitor and saturation with a finger or ear probe is useful for determining the degree of stress and hypoxia to which patients are subjected.

The purpose and nature of the test must be explained using standard phrases: 'The object of the test is for you to walk as far as possible in 6 minutes. Six minutes is a long time to walk, so you will be exerting yourself. You will probably get out of breath and be exhausted. You may even have to stop and rest. You may stop and rest, but must resume walking as soon as you are able.' And, 'You will walk back and forth around the course markers. You should pivot briskly around the markers and continue back the other way without hesitation.' Demonstrate how to walk and turn.

On **starting the test**, do not walk with the patient, but use standard phrases of encouragement, with an even tone of voice, each minute during the test: 'You are doing well, you have 5 minutes to go'. After 1 minute: 'Keep up the good work, you have 4 minutes to go', etc. The patient is permitted to rest, but the timer is not stopped. Instruct the subject to stop when 6 minutes are up, and measure the distance covered. Offer the patient a chair, and a drink of water. If the patient stops before the 6 minutes are up and refuses to continue (or you decide that they should not continue) record the distance. Repeat the Borg breathlessness scale.

Annexure B. Terminology and treatment of bronchial infections

The term 'chronic bronchitis' and its relationship to COPD and with other forms of bronchial infections is a source of confusion to many clinicians. The notes that follow, although not strictly within the brief of guidelines on COPD, are offered

Table VIII. Classification of bronchitis — diagnostic criteria, risk factors, usual pathogens and recommended treatment

Diagnosis	Criteria/risk factors	Usual pathogens	Recommended treatment*
1. Acute bronchitis	• Acute • No underlying lung disease	• Viruses	• Symptomatic • No antibiotic
2. Simple chronic bronchitis	• Symptoms define chronic bronchitis • Recent increase in sputum volume and purulence • FEV_1 > 60% predicted	• *H. influenzae* • *M. catarrhalis* • *S. pneumoniae* • Beta-lactam resistance possible	• Aminopenicillin • Amoxy-clavulinic acid • Macrolide/azalide
3. Complicated chronic bronchitis	• COPD with FEV_1 < 60% • Advanced age • ≥ 4 exacerbations/year • Recent increase in sputum volume and purulence • Significant morbidity (ill)	• Same organisms as in 2, but beta-lactam resistance more common • Sputum Gram stain and culture advised	• Amoxycillin-clavulinic acid • Second or third generation cephalosporin • New generation fluoroquinolone
4. Complicated chronic bronchitis with chronic bronchial sepsis	• As above with purulent sputum for long periods in year • + X-ray evidence of structural lung disease	• Same as 3, but also • Enterobacteriaciae • *Pseudomonas aeruginosa* • Sputum Gram stain and culture advised	• Ciprofloxacin or • Other quinolone • Alternative based on result of sputum culture

*Selection of antibiotic is influenced by knowledge of antibiotic resistance patterns in different areas of the country.

as a guide to assist clinicians to correctly assess and classify these conditions and offer appropriate treatment.

Chronic bronchitis. Chronic bronchitis is a condition identified on the basis of history — chronic cough with sputum production that develops in smokers and less commonly in persons with years of exposure to heavy atmospheric pollution. The cough typically begins as a 'smoker's cough', worse in the morning and productive of sputum which does not appear infected. It usually starts after a bout of winter bronchitis but persists for months and recurs the following year under similar circumstances. The British MRC defines chronic bronchitis as chronic productive cough that persists for at least 3 months of the year for 2 successive years. The pathology of the airways includes mucosal thickening with hypertrophy of mucous glands indicating mucus hypersecretion. Treatment is smoking cessation, which is usually associated with partial (rarely complete) improvement. Although chronic bronchitis is usually associated with airflow obstruction (as in patients with COPD), it can occur alone.

Classification of bronchitis

The classification of bronchitis and bronchial infections is difficult because of the plethora of terms and paucity of distinctive features. It is, however, important to classify disease correctly, as this serves as a guide to treatment. The following classification is recommended for use in South Africa (Table VIII).

- **Acute bronchitis:** Tracheobronchitis involving both the trachea and upper airways and caused by viruses.
- **Simple chronic bronchitis:** Chronic bronchitis as defined above, with normal or at most, mild impairment of lung function, with bronchitis unlikely to be complicated by respiratory failure.
- **Complicated chronic bronchitis:** Complications are severe airflow obstruction (< 50% of predicted), frequent exacerbations and/or severe respiratory limitation of activities.
- **Complicated chronic bronchitis with chronic bronchial sepsis:** Defined as infected sputum continuously or almost continuously throughout the year. This category includes patients with structural bronchial damage and even bronchiectasis from any cause.

Associated risk factors, causative pathogens and recommended antibiotic treatment are shown in Table VIII.

CLINICAL GUIDELINE

Guideline for Office Spirometry in Adults, 2004

South African Thoracic Society Standards of Spirometry Committee: E M van Schalkwyk, C Schultz, J R Joubert, N W White

Objective. To provide clinical guidelines for office spirometry in South Africa.
Options. More stringent guidelines are required for diagnostic laboratories and research.
Outcomes. To minimise variations in standard practice and improve the quality and usefulness of spirometry in the clinical setting.
Evidence. Recommendations are based on key international publications as well as research publications regarding reference values for South Africans.
Benefits, harm and costs. The medical, social and economic benefits and costs of standardisation of office spirometry in South Africa were considered in the recommendations.
Validation. The document has been reviewed and endorsed by the South African Thoracic Society.
Conclusions. The indications for spirometry must be specific and clear. Spirometry equipment must meet internationally accepted performance standards and carry proof of validation. Equipment must be regularly calibrated and maintained. Individuals performing spirometry must be adequately trained and demonstrate a high level of competence. Subject preparation, testing and quality control of results must be carried out according to published guidelines. Finally, test results must be interpreted according to current diagnostic guidelines, taking into account the purpose of the test, appropriateness of reference values and the clinical evaluation.

S Afr Med J 2004; **94:** 576-587.

1. Abbreviations

ATPS = ambient temperature, ambient pressure, saturated with water vapour; ATS = American Thoracic Society; BTPS = body temperature, ambient pressure, saturated with water vapour; ECSC = European Community for Steel and Coal; ERS = European Respiratory Society; FEV_1 = forced expiratory volume in 1 second; FVC = forced vital capacity; LLN = lower limit of normal; PEF = peak expiratory flow; RSD = residual standard deviation; SATS = South African Thoracic Society; TLC = total lung capacity; VC = vital capacity.

2. Introduction

Spirometry is an essential part of a complete respiratory evaluation, but inadequate standards and variations in standard operating procedures exist that reduce its clinical usefulness.[1] Good quality spirometry necessitates a competent operator, accurate and reliable equipment and a co-operative patient. Furthermore, it involves a series of standard procedures and quality control checks to produce technically satisfactory results. Finally, the results take reference standards into account and are interpreted with consideration of the clinical indications for testing.

Various authorities have published comprehensive guidelines for the standardisation of spirometry.[2-4] More recently, selective South African reference standards have become available for the normal range of forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV_1).[5-10] This statement is prompted by increased utilisation of office spirometry in South Africa and a perceived need for simplified guidelines for use at primary contact level, i.e. in the clinic or practice. Diagnostic and research lung function laboratories will require more comprehensive guidelines than proposed in this document.

Corresponding author: **Dr E M van Schalkwyk, Department of Medicine, Stellenbosch University, PO Box 19063, Tygerberg, 7505, e-mail emvs@sun.ac.za**

3. Definitions

Spirometry. Spirometry is one of a number of tests to evaluate respiratory function. The basic spirometric procedure involves the measurement of gas volume and rate of airflow during a maximal, forced expiration. The mechanical properties of the airways, lung, pleura, chest wall and respiratory muscles all contribute to these results.

Spirometer. Spirometers operate on one of two principles:

- Volume-type spirometers determine volume directly and have the advantages of low cost and ease of operation. However, data processing and storage capacity may be limited, unless the spirometer contains a microprocessor.
- Flow-type spirometers make use of a flow-sensor (pneumotach) to derive volumes. They are computerised, provide quick reference values, produce flow-volume loops enabling instant pattern recognition and can usually store

large data sets. On the other hand, they require greater expertise to operate, calibrate and maintain.

Spirogram. Spirograms are the graphic displays produced by spirometers. In addition to graphs, they provide the measured values (observed), the reference values (predicted) and the measured values expressed as a percentage of the reference values (% predicted). Volume-type devices generate volume-time curves (Fig. 1a) and flow-type devices generate flow-volume curves (Fig. 1b). Newer flow-type spirometers can produce both types of curve.

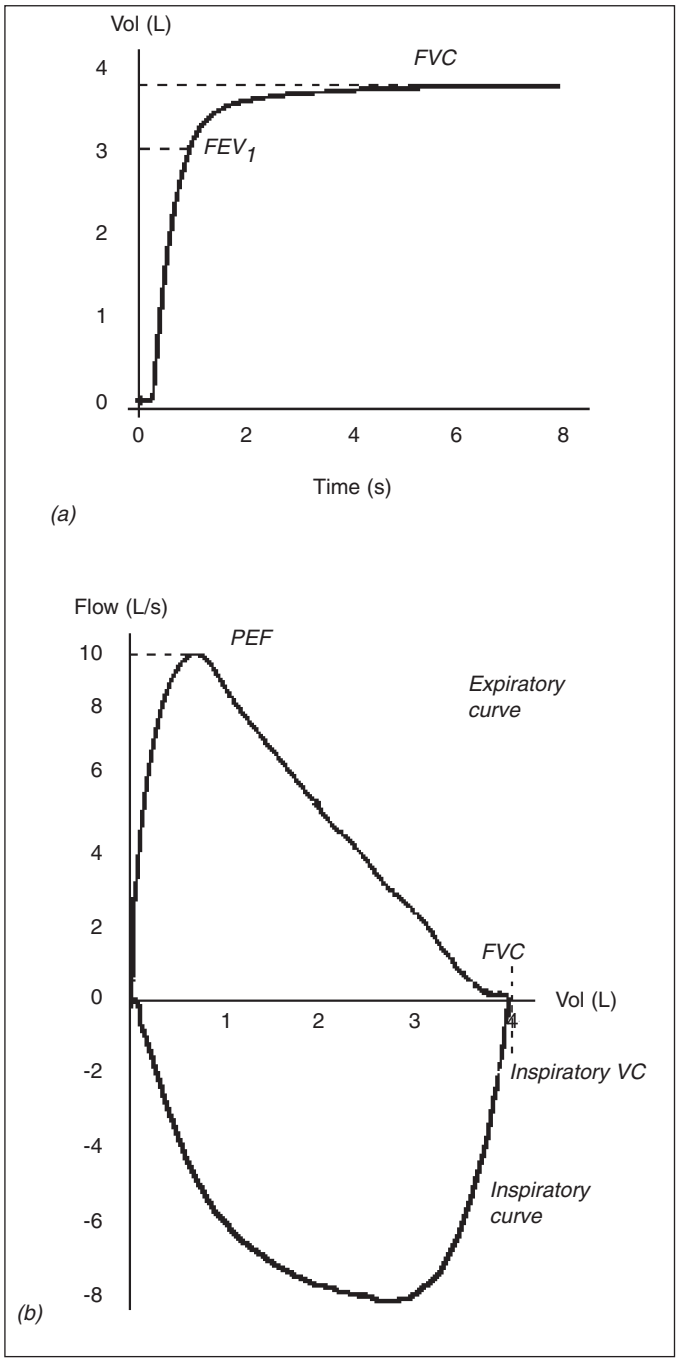

Fig. 1. (a) Volume-time, and (b) flow-volume curves. In the flow-type spirometer FEV_1 is a derived value. It can only be read from the flow-volume graph if a 1-second timer is displayed.

Measurements. Depending on type and level of sophistication, spirometers can produce a range of measurements that may assist in the clinical interpretation of results:

- *Vital capacity (VC)*: VC is the total volume of gas inhaled from the position of maximal expiration or exhaled from the position of maximal inspiration. It is measured with a relaxed/slow breathing manoeuvre either during inspiration or expiration. VC is expressed in litres (BTPS). BTPS refers to a standardised volume at normal body temperature (37°C) at ambient pressure, saturated with water vapour.
- *Forced vital capacity (FVC)*: FVC is the maximum volume of gas exhaled from the position of maximal inspiration by means of a rapid, maximally forced expiratory effort, expressed in litres (BTPS).
- *Forced expiratory volume in 1 second (FEV_1)*: FEV_1 is the volume of gas exhaled during the first second of the FVC manoeuvre, expressed in litres (BTPS).
- *FEV_1/FVC%*: FEV_1/FVC% is observed FEV_1 expressed as per cent of observed FVC ($FEV_1/FVC \times 100$).
- *Peak expiratory flow (PEF)*: PEF is the maximum flow generated with a FVC manoeuvre, expressed in litres per second (BTPS).

Measurements of FVC, FEV_1 and FEV_1/FVC% are the minimum required for diagnostic interpretation of results. VC measurements are useful for evaluating dynamic collapse of small airways as found in emphysema.

Calibration. Calibration is the process whereby the accuracy (truthfulness) and precision (repeatability) of a device such as a spirometer are tested and corrected using a gold standard such as a calibration syringe with a standard volume.

Validation. Validation is the process of establishing and certifying the accuracy and precision of a device.

Operator. The term operator refers to the person performing spirometry.

4. Indications for spirometry

Specific and clear indications for spirometry are helpful in the interpretation of results. The most frequent clinical indications for spirometry are listed below:

- To confirm a diagnosis in:
  - Individuals with suspected obstructive or restrictive lung disease.
- To grade respiratory impairment in.
  - Medico-legal cases (e.g. assurance or disability)
  - Individuals on treatment action plans (e.g. COPD)
  - Individuals for lung resection, and individuals for thoracotomy or upper-abdominal surgery if they have chronic respiratory diseases.

- To monitor changes in lung function in:
  - Individuals with chronic respiratory diseases — to evaluate responses to treatment and disease progression.
  - Workers regularly exposed to substances known to cause respiratory diseases.[11]
- To screen for lung disease in:
  - Smokers
  - Individuals with persistent respiratory symptoms, including shortness of breath (dyspnoea), chest tightness, wheezing, coughing, sputum production and chest pain.
  - New employees with potential for exposure to substances known to cause respiratory diseases — to determine baseline lung function.
  - Workers with significant exposure to substances known to cause respiratory diseases.

Spirometry is frequently applied in the occupational environment for surveillance purposes. Its sensitivity and specificity for detecting early disease varies and a screening programme should be tapered to the specific needs of the workplace. For example, early changes of COPD or asbestosis are detectable with spirometry, whereas early changes of silicosis are better detected with chest radiography. For occupational asthma, because of its varying nature, a respiratory symptoms questionnaire is frequently combined with spirometry in screening or surveillance programmes.

5. Specifications for spirometers

5.1 Proof of validation

Spirometers may lack accuracy and precision. Prospective purchasers of equipment should seek its proof of validation. Accuracy depends on the resolution (minimal detectable volume or flow) and linearity (consistency) of the entire system from the measuring components to the recording and display components. The American Thoracic Society (ATS) has published minimal performance criteria for *diagnostic* and *monitoring* spirometers and guidelines for validating equipment using waveform-generated calibration syringes.[2] Selective ATS recommendations for *diagnostic* spirometers are provided in Tables I and II. Table II provides standards for graph output. Manufacturers should follow these guidelines to ensure that spirometers provide accurate data that are comparable between different settings and over time. Commercially available devices for *monitoring* of FEV_1 and PEF have disadvantages for office spirometry because they may be less accurate, usually cannot be calibrated to ensure their performance, and graphical displays may be absent or inadequate for evaluation of test quality.

Other recommendations include:

- The BTPS-correction facility that meets ATS standards: The volume of exhaled gas is measured outside the body at ambient conditions, designated ATPS (ambient temperature, ambient pressure, saturated with water vapour). These gas measurements are corrected to reflect conditions inside the lung (BTPS). Without this facility, mathematical correction of volumes has to be done manually.[2]

Table II. Minimum scale factors for spirograms*

Parameter	Required resolution	Scaling
Volume	0.025 l	10 mm/l
Flow	0.1 l/s	5 mm/l/s
Time	0.2 s	2 cm/s

*For the flow-volume curve exhaled flow is plotted upwards and exhaled volume towards the right in a 2:1 ratio.

Table I. Selective minimum volume and flow criteria for diagnostic spirometers

Parameter	Required range	Accuracy (BTPS)	Flow range (l/s)	Time (s)	Validation method
VC	0.5 - 8 l	± 3% of reading or ± 0.050 l, whichever is greater	0 - 14	30	3 l calibrated syringe
FVC	0.5 - 8 l	± 3% of reading or ± 0.050 l, whichever is greater	0 - 14	15	24 standard waveforms/ 3 l calibrated syringe
FEV_1	0.5 - 8 l	± 3% of reading or ± 0.050 l, whichever is greater	0 - 14	1	24 standard waveforms
PEF		± 10% of reading or ± 0.400 l/s, whichever is greater Precision: ± 5% of reading or ± 0.200 l/s, whichever is greater	0 - 14		26 flow standard waveforms

- Facility to generate real-time spirograms — to enhance feedback and subject compliance.
- Stated source(s) of reference values and facility to select or enter appropriate values manually.
- Computer-driven technical quality indicators that meet ATS standards (computer automatically evaluates test quality based on pre-programmed criteria and gives prompts).
- Printing facility for record-keeping purposes.
- Adequate facility to save large numbers of tests and test quality indicators where needed, for example, for occupational surveillance.
- Availability of after-sales service.

In addition to mechanical validation, spirometers can also be tested in real-life situations involving human subjects.[12]

SATS recommend that independent professional advice from a registered pulmonology training laboratory or the Spirometry Training and Certification Committee of the South African Thoracic Society (SATS) be obtained before a new spirometer is acquired.

5.2 Calibration

All diagnostic spirometers must be volume-calibrated at least daily using a calibrated syringe with a volume of at least 3 l to ensure that they remain accurate during use. During industrial surveys in which a large number of subject manoeuvres are performed, calibration must be checked each morning and at least twice during the day. In circumstances where the temperature may change markedly over the day, for example in field surveys, more frequent temperature corrections are necessary.

Calibration involves the following steps:

1. The spirometer is switched to calibration mode (to prevent BTPS-correction because room air is injected). Room temperature and barometric pressure readings are entered. In the absence of a barometer, barometric pressure readings can be obtained from the local airport or weather bureau.

2. Calibration syringe size is specified. A 3 l syringe is recommended. Currently, the use of 2 l and 1 l syringes is not validated.

3. The calibration syringe is connected to the spirometer and the maximum volume of air injected. Flow-type spirometers are calibrated by injection of the maximum volume from the syringe at least three times, each time at a different speed, to cover a range of flow rates. Calibration is complete when the recorded volumes are within 3% or 50 ml, whichever is the greater, for each flow rate tested. In the event of in-line (antimicrobial) filters being used, calibration should be done with a filter installed. The quality of the filter must be such that the spirometry system still meets ATS standards.

4. Volume-type spirometers are checked for air leaks if the measured volume remains outside the acceptable range. A leak can be detected by applying a slight constant positive pressure with the calibration syringe while the spirometer outlet is occluded. Any volume change greater than 10 ml after 1 minute indicates a leak. Faults are corrected and calibration repeated.

5. Remaining problems are logged and referred to the manufacturer without delay.

The use of biological standards such as, for example, the operator for daily volume calibration (biological calibration) cannot replace the use of a calibration syringe. Lung function testing involves a 'system' consisting of three main components: spirometer, operator and test subject. Each of these can be a source of variation in measurements and syringe calibration is required in order to isolate the device. Biological standards are useful for testing software irregularities such as, for example, inconsistencies in the calculation of predicted values. Also, when they are used in conjunction with a physical standard (calibration syringe), biological standards are useful to test the proficiency of operators.

In addition to daily volume calibration, spirometers must be maintained routinely according to the manufacturer's specifications. This includes the cleaning of pneumotachs at least once a week (more frequently if there is visible condensation), as they are particularly sensitive to moisture and secretions. Other components of the spirometer, for example the time clock, must also be calibrated from time to time. For these and other maintenance functions the manufacturer must routinely check spirometers at least 6 - 12-monthly.

6. Responsibilities of operators

6.1 Skills

Operators must have an understanding of the principles underlying the measurement and equipment operation. They must also be able to ensure optimal subject co-operation, provide acceptable, reproducible results and recognise common abnormalities. Training of pulmonary medical technologists includes this competency and competency to perform advanced lung function tests and laboratory quality assurance. The SATS is in the process of developing a curriculum, training materials and a means of certification of proficiency in performing spirometry for people other than pulmonary medical technologists.

6.2 Quality assurance

A quality assurance programme is critical to ensure a well-functioning spirometry laboratory.[13] This may be difficult to attain in a routine clinical practice. At a minimum, a calibration and maintenance log as well as electronic or hard copies of whole spirograms must be kept so that accuracy and precision of past tests can be verified. Additionally, standard operating procedures should be documented and kept for reference purposes.

6.3 Infection control

Various components of the spirometry system, including mouthpieces, nose clips, pneumotachs, valves and tubing, are potential vehicles for transmission of infection to subjects and staff. Transmission of upper respiratory tract infections, enteric infections and blood-borne infections such as hepatitis and HIV, can potentially occur through direct contact when test subjects have open sores in the mouth, bleeding gums or haemoptysis. Tuberculosis and viral and nosocomial infections can also occur, indirectly, through inhalation of aerosol droplets from the spirometer or surroundings. The type of test manoeuvre determines whether inhalation from the spirometer takes place. This has a major influence on the extent of infection control needed. An expiratory manoeuvre without inhalation from the spirometer reduces the potential for cross-infection dramatically and is the method of choice for mass screening purposes.

Infection control recommendations for expiratory manoeuvres without inhalation from the spirometer:

- Spirometry should be performed in a well-lit and ventilated area.
- Hands must be washed immediately after direct handling of mouthpieces or other potentially contaminated spirometer parts, and between subjects, to avoid operator exposure and cross-contamination. Gloves should be worn for personal protection if there are open cuts or sores on the operator's hands.
- A clean disposable mouthpiece or a disinfected re-usable mouthpiece must be used for every test subject. Any other spirometer part coming into direct contact with mucosal surfaces must be decontaminated/sterilised.
- Spirometers must be cleaned regularly according to the manufacturer's recommendations and the frequency of tests done. Any part with visible condensation from expired air must also be decontaminated before re-use.

Additional infection control recommendations for manoeuvres involving inhalation from the spirometer system or part of the system:

- In-line filters must be used and replaced after each subject, or
- Involved parts of the system (i.e. spirometer, breathing tubes and resistive element of the pneumotach) must be decontaminated/sterilised/flushed after each subject. (Note: re-calibration is necessary every time a system has been dismantled for decontamination.)

Special precautions for patients with haemoptysis or known transmissible infections such as tuberculosis:

- In-line filters must be used routinely (even if expiratory manoeuvres are performed exclusively) with sterilisation of contaminated surfaces only, or
- Equipment must be decontaminated/sterilised/flushed completely after each case. Testing such cases at the end of the day will allow for overnight decontamination of equipment. (Note: indications for spirometry in known active tuberculosis are limited.)

For decontamination/sterilisation procedures, consult the user manual or contact the infection control unit or lung function laboratory at an academic hospital near you.

7. Preparation of subjects

7.1 Exclusion criteria

The main exclusion criterion for spirometry in routine clinical practice is current respiratory infections in individuals for impairment/disability assessment. Respiratory infections can cause temporary lung function impairment and spirometry, if required, should be done only once infections, including tuberculosis, have resolved.

7.2 Personal information

The following information is required for reference purposes (section 9.2) and must be entered into the programme: weight and standing height, age, sex and race. For height and weight measurements the subject should be barefoot and wear only light clothing. It is also useful for interpretation purposes to record the time of last bronchodilator use and smoking status.

7.3 Positioning and preparation

The subject must be made to feel comfortable. Shelter him/her from other subjects to minimise inhibitions or distractions. Loosen tight clothing. Leave well-fitting dentures in, but remove loose-fitting ones. Test the subject sitting upright on a firm chair with his/her chin slightly elevated and neck slightly extended. This posture should be maintained during the forced expiration. Discourage excessive bending at the waist. Use of a nose clip is strongly recommended. Instruct the patient when to insert the mouthpiece, for example, at the end of maximal inspiration. Ensure that the subject does not bite the mouthpiece too hard, that the lips are sealed tightly around it, and that the tongue does not obstruct the mouthpiece in any way.

Ensure maximum subject co-operation. Submaximal efforts are a frequent cause of abnormal results. Explain techniques in simple terms and demonstrate them to the patient. For example, explain that: 'I am going to have you blow into the machine to see how big your lungs are and how fast the air comes out. It does not hurt but requires your co-operation and lots of effort.' Explain and demonstrate the use of a nose clip and mouthpiece. Remind the patient of a few key points. 'Be sure to take as deep a breath as possible, blast out hard and do not stop blowing until I tell you to do so.' Give feedback about the performance, encourage and describe what improvements can be made.

8. Execution of tests

8.1 Test manoeuvres

Test manoeuvres are determined in part by the setting and level of sophistication of the spirometer:

- **Expiratory-only method.** For reasons of ease, cost and infection control this method is recommended for mass screening. It consists of a FVC test with or without a slow VC test. For the FVC test, the test subject is required to inhale maximally before inserting the mouthpiece and starting the test. Expiration must be rapid, forceful and complete, lasting at least 6 seconds. If significant obstruction is demonstrated, proceed with a slow VC test. The slow VC test is preceded by a maximal inspiration, the mouthpiece is inserted and the patient then breathes out in a relaxed fashion and for as long as possible. Allow for up to 15 seconds. Only the VC is recorded. *The rationale for performing a slow VC test is as follows: the slow VC provides additional information on the characteristics of the obstructive defect. A reduction in FVC compared with slow VC suggests dynamic collapse of unsupported airways during forced expiration leading to air trapping. This pattern is typically seen in emphysema.*
- **Inspiratory-expiratory method.** With this method both inspiration and expiration are recorded to generate a flow-volume curve on a flow-type spirometer. Typically, after insertion of the mouthpiece, a period of quiet breathing is followed by a complete expiration, a rapid, forceful and complete inspiration and finally, a rapid, forceful and complete expiration. Some programmes prompt for an expiratory manoeuvre followed by an inspiratory manoeuvre. However, the first method is recommended, because this will reveal air trapping as described in the previous section. Reduced FVC compared with forced, inspiratory VC is suggestive of air trapping.

8.2 Test quality

The final step in ensuring data quality is the evaluation of spirograms for *acceptability* and *reproducibility.*

8.2.1 Acceptability

A technically acceptable FVC trial (Fig. 1) must exhibit the following qualities:

- A 'crisp', unhesitating start.
- PEF of the flow-volume curve achieved within the first 25% of the volume expired from maximum inspiration. (Most individuals are able to produce PEF within the first 15% of the volume expired.)
- A continuous smooth exhalation without artefacts caused by coughing, variable effort, second inhalations or leaks influencing FEV_1 or FVC.
- A complete exhalation (to the point where no more air can be expelled from the lungs), lasting until the volume-time curve has clearly reached a plateau or the flow-volume curve has progressively returned to zero flow.

All technically unsatisfactory trials must be rejected. Common patterns are illustrated in Figs. 2 - 4.

8.2.2 Reproducibility

Suboptimal effort by the test subject is a frequent cause of diminished lung function results. Ensuring reproducibility of test results is a way of verifying that the test subject co-operates fully and provides maximal effort. Reproducibility is defined as two curves in which the difference in FVC and FEV_1, respectively, do not exceed 0.2 l. Reproducibility is usually evident from the spirogram at a glance (Fig. 5). Testing

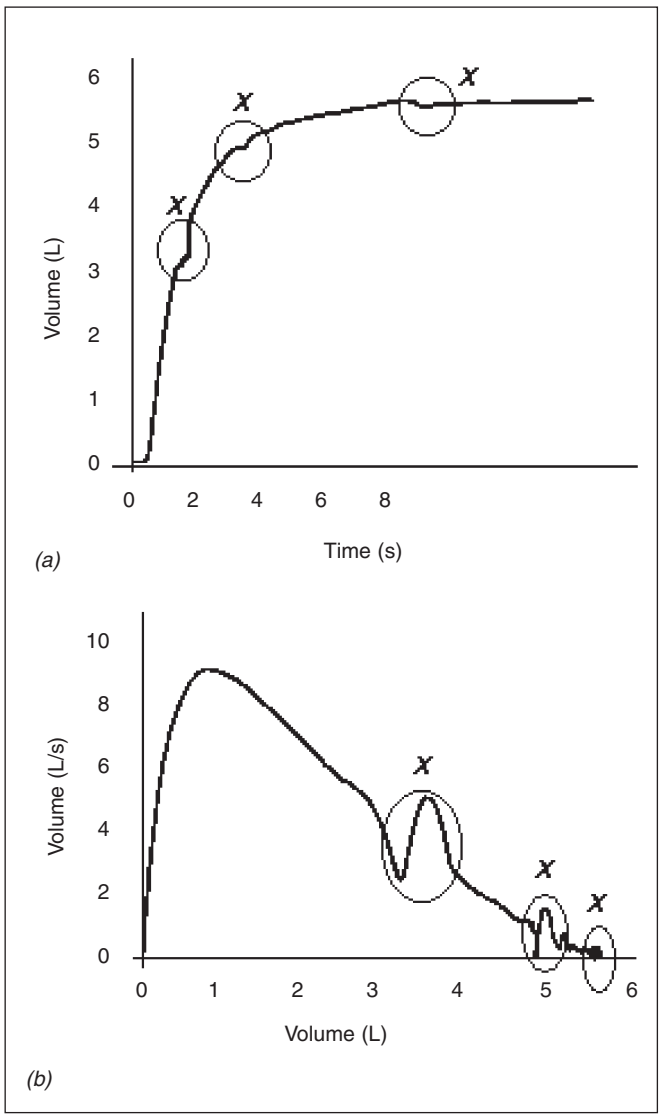

Fig. 2. (a) Volume-time, and (b) flow-volume curves exhibiting cough artefacts (X) that can influence observed FVC and FEV_1. Volume-time graphs are better for evaluating end-of-test quality.

must continue until a minimum of three technically *acceptable* FVC trials have been obtained, at least two of which are *reproducible*. However, no more than eight trials should be performed during a single session, because fatigue induced by repeated FVC trials can lead to reduced results. Subjects with asthma sometimes demonstrate spirometry-induced bronchoconstriction leading to a progressive reduction in lung function with successive trials. This finding will be of interest to the clinician and all acceptable curves should be kept for reporting. Failure to obtain reproducibility after eight trials must be documented, but selection of the best curve may proceed.

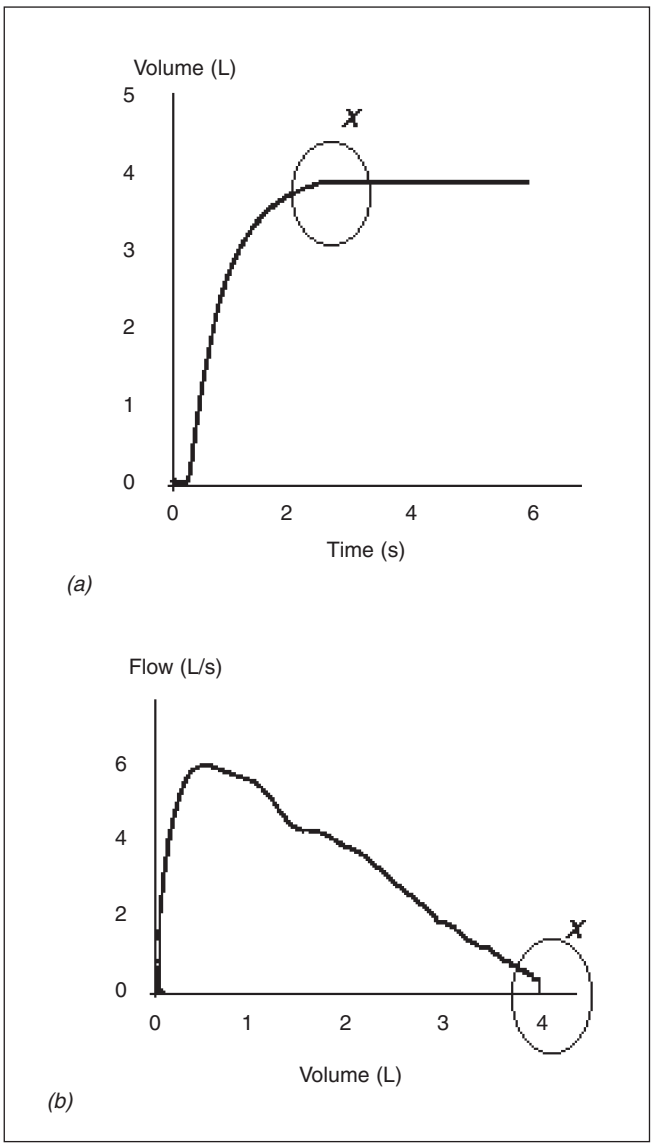

Fig. 3. (a) Volume-time, and (b) flow-volume curves exhibiting glottis closure (X) resulting in premature termination of effort and reduced observed FVC.

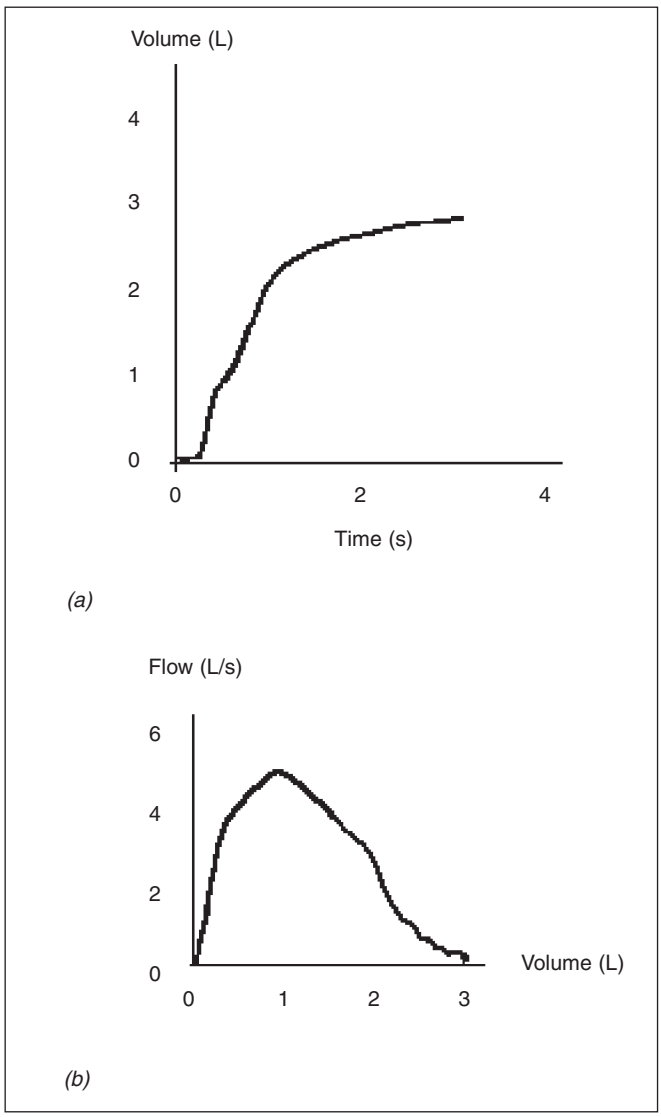

Fig. 4. (a) Volume-time curve exhibiting a slow rise and the end-of-test not reaching a plateau, and (b) flow-volume curve with a late peak flow and an abrupt end-of-test. Failure to demonstrate reproducibility will confirm these as submaximal efforts.

9. Interpretation of results

9.1 Selection of the best test

For diagnostic purposes, the best spirogram must be inspected, i. e. the graph with the largest sum of FVC and FEV_1. For impairment or severity grading the highest values recorded for FVC and FEV_1 must be selected from all acceptable curves, including the post bronchodilator curves, even if they come from separate curves.

9.2 Reference standards

An individual's observed results are evaluated for abnormalities against predicted results derived from a normal

reference population. The comparison is made as per cent observed/predicted. Predicted values for FVC and FEV_1 are calculated from equations based on age, height and gender because these characteristics are the most important determinants of lung and airway size in healthy individuals.[14-17] Office spirometers are typically programmed with prediction equations derived from the study of Caucasians, such as the European Community for Steel and Coal (ECSC) (Table III).[17] Caucasians, when compared with indigenous populations, usually show higher FVC and FEV_1, but similar or lower FEV_1/FVC%. The use of inappropriate predicted values can result in an increased rate of abnormal results in clinically normal people.

The use of prediction equations based on studies carried out in South Africa, has been investigated.[18] Indigenous equations, as detailed in Table IV, are, where available, recommended for population screening, surveillance and medico-legal purposes. However, it is acknowledged that the application of these predicted values in every context where spirometry is used may present practical difficulties. Alternatively, office spirometers usually have a facility for application of a correction factor such as 0.9 for adjusting predicted values for Caucasians with a view to their being used for indigenous populations. Adjusted per cent predicted can be calculated using the formula:

$$[\text{Observed}_{\text{indigenous}} / (\text{predicted}_{\text{Caucasian}} \times 0.9)] \times 100]$$

While the use of such correction factors is acceptable, when understood as an approximation, use of the recommended prediction equations is the preferred option. Operators must familiarise themselves with their spirometers regarding these conditions.

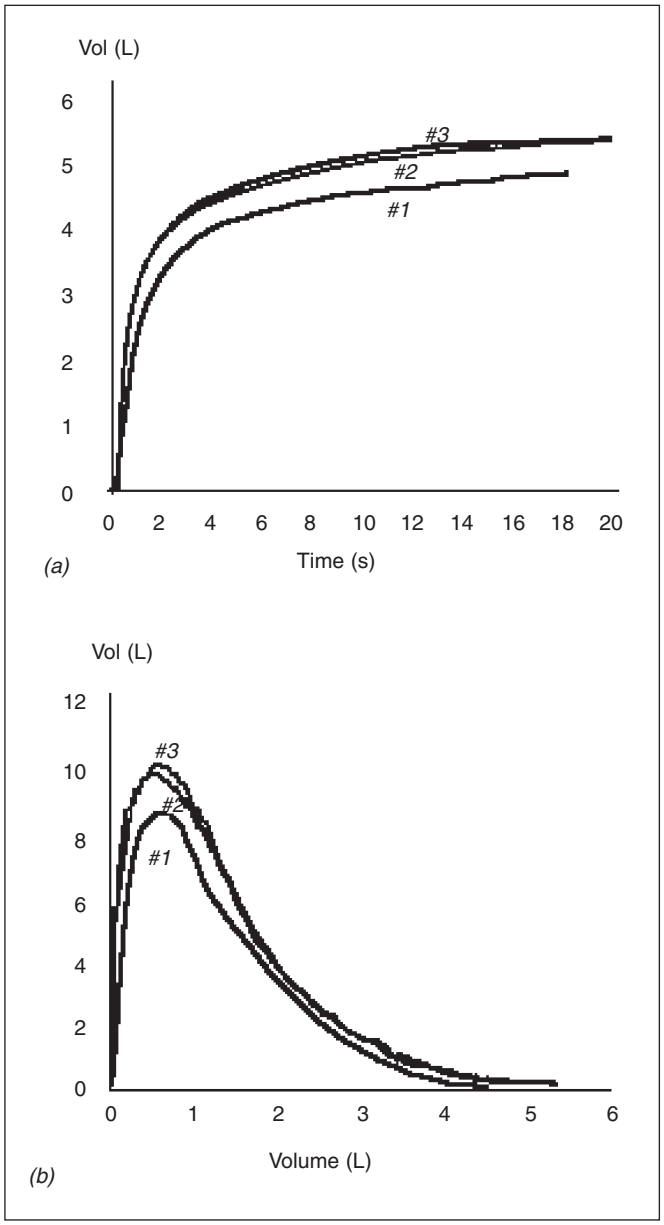

Fig. 5. (a) Volume-time, and (b) flow-volume curves each demonstrating three acceptable FVC trials, only #2 and #3 of which are reproducible.

Table III. ECSC prediction equations* from Quanjer *et al.*[17]

Parameter	Prediction equation	1.64 × RSD
Men		
FEV_1† (l)	4.30H – 0.029A – 2.49	0.84
FVC† (l)	5.76H – 0.026A – 4.34	1.00
FEV_1/VC%	– 0.18A + 87.21	11.8
Women		
FEV_1† (l)	3.95H – 0.025A – 2.60	0.62
FVC† (l)	4.43H – 0.026A – 2.89	0.71
FEV_1/FVC%	– 0.19A + 89.10	10.7

*Valid for age 18 - 70 years. Between age 18 and 25 years substitute age 25 in the equation. The lower limit of normal (LLN) is the lower 5th percentile: predicted value – 1.64 × RSD.
†80% predicted is an accepted alternative LLN.
H = standing height (m); A = age (years); RSD = residual standard deviation.

Table IV. Prediction equations* from Louw *et al.*[5] (African men) and Mokoetle *et al.*[8] (African women)

Parameter	Prediction equation	1.64 × RSD
Men		
FEV_1 (l)	2.9H – 0.027A – 0.54	0.75
FVC (l)	4.8H – 0.024A – 3.08	0.89
Women		
FEV_1 (l)	3.4H – 0.028A – 1.87	0.64
FVC (l)	4.5H – 0.023A – 3.04	0.67

*The lower limit of normal (LLN) is the lower 5th percentile: predicted value – 1.64 × RSD. 80% is an acceptable alternative LLN.
H= standing height (m); A = age (years); RSD = residual standard deviation.

9.3 Diagnosis and severity grade

9.3.1 Algorithm

The major aims of interpreting spirometric results are to confirm the clinical diagnosis and to estimate the severity of the disease. An algorithm is presented (Fig. 6) for categorising spirometric results as obstructive, normal or restrictive patterns. The algorithm employs three variables, namely FEV1/FVC%, % predicted FVC and % predicted FEV_1. The interpretative strategy proposed is based on published guidelines,[19] but the lower limit of normal (LLN) for FEV_1/FVC% has been adapted to conform to current diagnostic guidelines for chronic obstructive pulmonary disease (COPD).[20]

The LLN for FEV_1/FVC%, FVC and FEV_1, is the 5th percentile (see Tables III and IV). Eighty per cent predicted is an acceptable alternative LLN for FVC and FEV_1. The use of a fixed percentage for the LLN for FEV_1/FVC% (usually 70% or 75%) is a pragmatic clinical approach, but has limitations. For screening purposes it may be more accurate to use the 5th percentile to minimise misclassifications of borderline values. A reduced FEV_1 should always be regarded as abnormal. When this is the only finding on the spirogram, further investigations, including a bronchodilator test, may be necessary to define the abnormality (see Fig. 6).

Non-clinicians such as, for example, occupational health nurses can use the algorithm to identify cases for referral. The experienced clinician will use this information in combination with pre-test information, including the indications for testing, and his/her knowledge about the case to make a final clinical diagnosis.

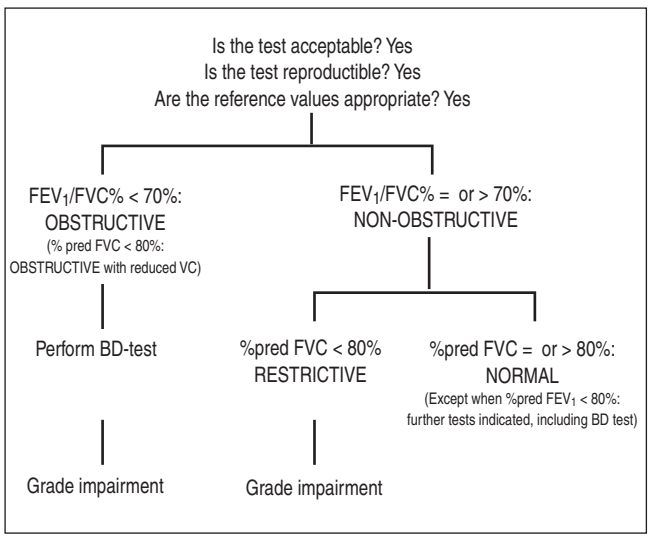

Fig. 6. An algorithm for categorising spirometric results is presented. The observed FEV_1/FVC is expressed as a percentage and the lower limit of normal (LLN) is defined as 70%. FVC and FEV_1 are based on per cent predicted (%pred) and LLN defined as 80% (BD = bronchodilator).

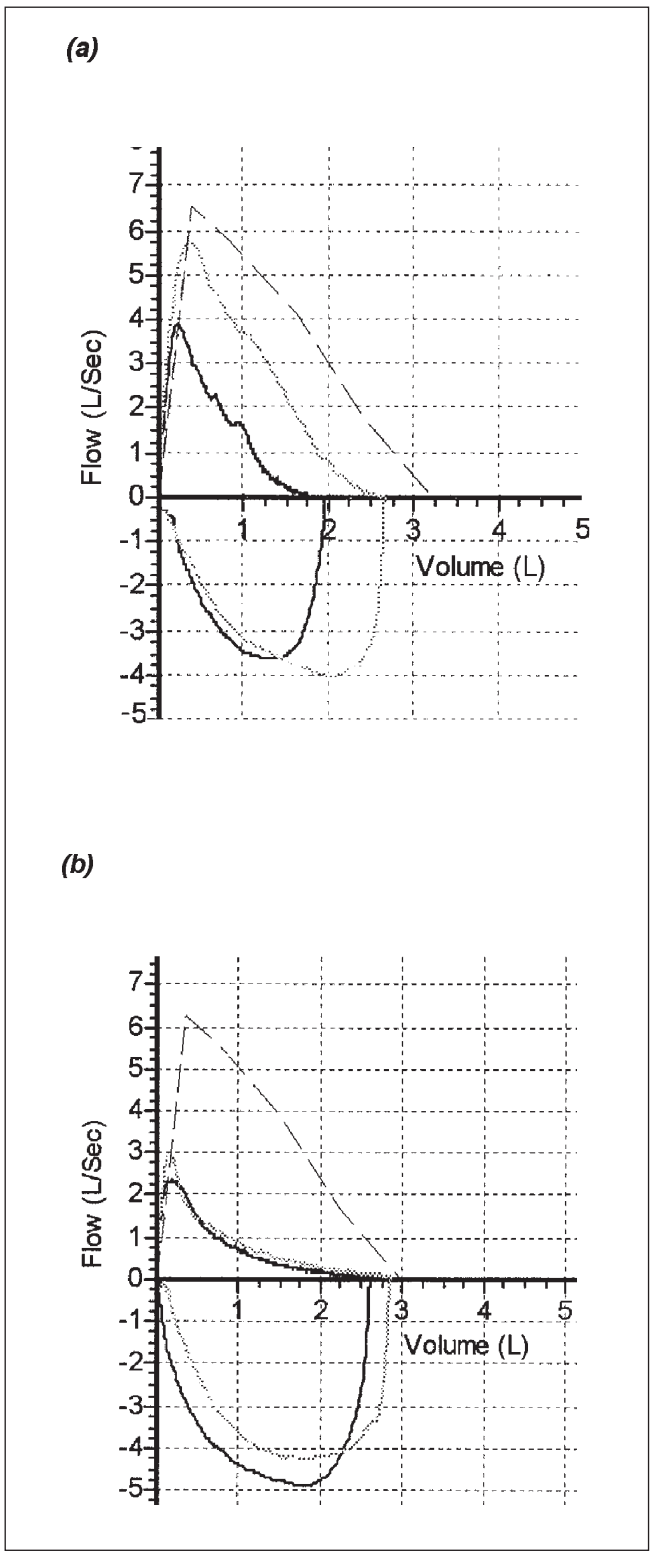

Fig. 7. Flow-volume curves exhibiting typical (a) reversible obstruction in an asthmatic, and (b) non-reversible obstruction in a person with COPD (black = pre-bronchodilator, grey = post-bronchodilator, broken line = reference standard).

9.3.2 Obstructive defect

An obstructive ventilatory defect is defined as a disproportionate reduction in maximal airflow from the lung with respect to the maximal volume that can be displaced from the lung. The experienced clinician will readily recognise a pattern of expiratory airflow limitation on the flow-volume curve (Fig. 7). The diagnosis of an obstructive defect should be followed up with a bronchodilator test to examine the nature of the obstruction. Severity of obstruction is graded according to the worst affected spirometric parameter, usually % predicted FEV_1. Mild obstructive defects could be missed if there is under-estimation of FVC due to unacceptable end-of-test criteria.

9.3.3 Restrictive defect

A restrictive ventilatory defect is characterised physiologically by a reduction in total lung capacity (TLC) as determined by advanced lung function testing. One may infer a restrictive defect when FEV_1/FVC% is normal or high (non-obstructive) and FVC is reduced (Fig. 8). The severity of the restrictive defect is graded according to TLC when available, otherwise it is graded according to the worst affected spirometric parameter, and usually % predicted FVC.

A range of conditions can reduce FVC *per se*:

- Conditions impeding movement of the chest wall (e.g. pain, pleural thickening or effusion, neuromuscular weakness, skeletal abnormality or hyperinflation with air trapping as found in COPD).
- Diffuse conditions of lung parenchyma causing stiffness of the lung (e.g. interstitial lung disease with fibrosis, pulmonary oedema).
- Conditions causing reduced communicating lung volume (e.g. lung resection, occlusion of a main bronchus, post-tuberculous lung destruction and space-occupying lesions in the chest).

Restrictive abnormalities are often over-diagnosed because of poor effort by the patient (Figs 3 and 4) or the use of inappropriate prediction equations. Nevertheless, diagnostic interpretation of a reduced FVC can be difficult and referral to a specialist must be considered after exclusion of obvious technical causes.

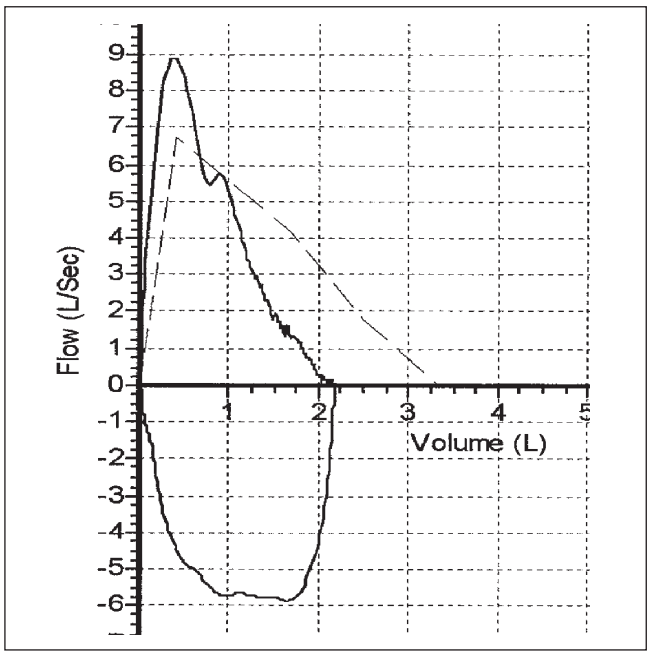

Fig. 8. Flow-volume curve exhibiting a typical restrictive pattern in a person with sarcoidosis (broken line = reference curve). The 'shoulder' on the down-slope of the expiratory curve was reproducible (not demonstrated). It represents a normal physiological phenomenon of the expiratory curve.

9.3.4 Obstruction with reduced FVC

This pattern consists of reduced FEV_1/FVC% and FVC and is usually found in obstructive conditions such as, for example, severe emphysema or asthma, but a combination of an obstructive and restrictive condition can produce a similar result. Other VC manoeuvres (section 8.1) and a bronchodilator test (Fig. 7), performed in the office, can assist in further defining the underlying disease. The severity of the defect is graded according to the indicator showing the most severe defect, usually % predicted FEV_1.

9.3.5 Bronchodilator response

The purpose of a bronchodilator test is to determine whether airway obstruction, as measured by spirometry, is reversible with inhaled beta-2 agonists (Fig. 7). A bronchodilator test can be standardised as follows:

1. Two reproducible FVC trials are obtained from the test subject.
2. Two puffs (400 µg) of salbutamol or equivalent are administered.
3. A waiting period of at least 10 minutes is introduced.
4. Two reproducible FVC trials are again obtained.
5. The best post-bronchodilator FEV_1 is evaluated for a significant improvement of at least 200 ml *and* 12% from the best pre-bronchodilator FEV_1. Per cent improvement in FEV_1 can be calculated using the formula:

$$[FEV_{1\ pre\text{-}BD} - FEV_{1\ post\text{-}BD} / FEV_{1\ pre\text{-}BD}] \times 100$$

The post-bronchodilator FVC trials must be done at least 10 minutes after administration of the bronchodilator, but ideally only after 20 - 30 minutes, as this is the time of maximum effect of most short-acting bronchodilators. Both the pre- and post-bronchodilator FEV_1 must be reproducible; otherwise a response cannot be confidently interpreted as such. For an accurate interpretation of a negative response, subjects must have been weaned from short-acting bronchodilators for at least 4 hours and long-acting bronchodilators and theophylline for at least 12 hours, if medically possible. A number of factors, including the dose of bronchodilator, recent prior bronchodilator medication and timing of the post-bronchodilator FVC trials can influence the magnitude of the

Table V. Guide for grading* spirometric results with a view to quantifying respiratory impairment

Parameter	Normal	Mild (able to meet physical demands of most jobs)	Moderate (diminished ability to meet physical demands of many jobs)	Severe (unable to meet physical demands of most jobs)
% pred FVC	≥ 80	60 - 79	51 - 59	≤ 50
% pred FEV_1	≥ 80	60 - 79	41 - 59	≤ 40
FEV_1/FVC%	≥ 70	60 - 69	41 - 59	≤ 40

*Impairment grade is allocated according to the worst affected parameter. Refer to a pulmonologist if impairment grade and clinical assessment do not agree.

response significantly. Each practice should decide on a standard protocol.

9.3.6 Grading respiratory disease severity

The main indications for grading respiratory disease severity are to quantify respiratory impairment/disability for medico-legal purposes, and to optimise and standardise treatment practices.

Guidelines for grading spirometric impairment correlate different lung function tests, including spirometry, with the ability to perform physical activities.[21] For this purpose criteria for spirometry, performed in the office, are included (Table V) for use in conjunction with the algorithm. LLN for FEV_1/FVC% has been adapted to conform to current diagnostic guidelines for chronic obstructive pulmonary disease (COPD).[20] A severity grade is awarded according to the worst affected parameter. The grading of obstruction should be based on the post-bronchodilator values.

In most cases simple spirometry will be sufficient for evaluating respiratory impairment. However, if discordance is found between spirometry and the stated level of dyspnoea or clinical evaluation, additional lung function tests may be indicated and the subject must be referred to a specialist with diagnostic lung function facilities. Further tests might include carbon monoxide diffusing capacity (DLCO) and/or exercise testing. In addition to spirometry, DLCO is clinically one of the most useful tests of lung function. It is especially useful in interstitial lung diseases, including the pneumoconioses, where gas transfer at alveolar level might be affected disproportionately to the mechanical properties of the lung. Another factor that needs to be considered during the clinical evaluation is the potential contribution of extra-pulmonary disease, for example, ischaemic heart disease, to total impairment. Also, because of its varying nature, the usual spirometric criteria do not apply to asthma as far as assessment of impairment/disability is concerned.[22]

As stated before, treatment guidelines also use spirometric grading to standardise treatment practices. These guidelines for grading severity are usually disease-specific and their main aims are to control the disease and improve prognosis. Therefore, the spirometric grading could differ from general guidelines aimed primarily at quantifying functional impairment.

9.4 Reporting

Spirometry reports must contain the following information:

- Identification of subject and date of testing.
- Personal information (see section 7.2) and origin of reference values.
- Numerical values and graphs to assess acceptability and reproducibility (at least two curves, but preferably three).
- Latest calibration date.

The report should refer to lung function and not disease (e.g. 'obstructive lung function defect without reversibility' rather than 'chronic obstructive lung disease'), unless the reporter is a clinician and has full clinical details to make an appropriate diagnosis.

10. Spirometry Training and Certification Committee

For further information on training opportunities, readers may contact the Chair, Spirometry and Training Certification Committee, South African Thoracic Society, PO Box 16433, Vlaeberg, 8018.

11. Acknowledgement

The Standards of Spirometry Committee of the SATS drafted this document. The SATS Council adopted it in August 2001. The working group wishes to thank all reviewers for their input and the staff of the lung function laboratory at Tygerberg Hospital for help with graphic material.

12. References

1. Basson E, Stewart RS. The standards of spirometry in the RSA. *S Afr Med J* 1991; **79:** 361-363.
2. American Thoracic Society. Standardization of spirometry. 1994 Update. *Am J Respir Crit Care Med* 1995; **152:** 1107-1136.
3. British Thoracic Society and Association of Respiratory Technicians and Physiologists. Guidelines for the measurement of respiratory functions. *Respir Med* 1994; **88:** 165-194.
4. Stewart RI, Basson E. Standardisation of spirometry. *S Afr Med J* 1991; **79:** 401-404.
5. Louw SJ, Golden JG, Joubert G. Spirometry of healthy adult South African men. Part I. Normative values. *S Afr Med J* 1996; **86:** 814-819.

6. Goldin JG, Louw SJ, Joubert G. Spirometry of healthy adult South African men. Part II. Interrelationship between socio-environmental factors and 'race' as determinants of spirometry. *S Afr Med J* 1996; **86:** 820-826.
7. Hnizdo E, Churchyard G, Dowdeswell R. Lung function prediction equations derived from healthy South African gold miners. *Occup Environ Med* 2000; **57**: 698-705.
8. Mokoetle K, De Beer M, Becklake MR. A respiratory health survey of a black Johannesburg workforce. *Thorax* 1994; **49:** 340-346.
9. White N, Hanley JH, Lalloo UG, Becklake MR. Review and analysis of variation between spirometric values reported in 29 studies of healthy African adults. *Am J Respir Crit Care Med* 1994; **150:** 348-355.
10. Lalloo UG. Respiratory health survey in an Indian South African community: Distribution and determinants of symptoms, diseases and lung function. MD thesis, University of Natal, 1992.
11. Ehrlich RI. Occupational medical surveillance. *South African Journal of Continuing Medical Education* 1996; **14:** 1301-1310.
12. Maree DM, Videler EA, Hallauer M, Pieper CH, Bolliger CT. Comparison of a new desktop spirometer (Diagnosa) with a laboratory spirometer. *Respiration* 2001; **68:** 400-404.
13. American Thoracic Society. Quality assurance in pulmonary function laboratories. *Am Rev Respir Dis* 1986; **134:** 625-627.
14. Yang T-S, Peat J, Keena V, Donnelly P, Unger W, Woolcock A. A review of the racial differences in the lung function of normal Caucasian, Chinese and Indian subjects. *Eur Respir J* 1991 ; **4:** 872-880.
15. Hankinson JL, Kinsley KB, Wagner GR. Comparison of spirometric reference values for Caucasian and African American blue-collar workers. *J Occup Environ Med* 1996; **38:** 137-143.
16. Knudson RJ, Slatin RC, Lebowitz MD, Burrows B. The maximal expiratory flow-volume curve. Normal standards, variability and effects of age. *Am Rev Respir Dis* 1976; **113:** 587-600.
17. Quanjer PH, Tammeling GJ, Cotes JE, Pederson OF, Peslin R, Yernault JC. Lung volumes and forced ventilatory flows. Report Working Party Standardization of Lung Function Tests, European Community for Steel and Coal. Official Statement of the European Respiratory Society. *Eur Respir J Suppl* 1993; **16:** 5-40.
18. Safety in Mines Research Advisory Committee. Ehrlich R, White N, Myers J, *et al*. Lung Function Reference Tables for Use in the South African Mining Industry. Health Report 610. May 7, 2000. www.simrac.co.za
19. American Thoracic Society. Lung function testing: selection of reference values and interpretative strategies. *Am Rev Respir Dis* 1991; **144:** 1202-1218.
20. Pauwels RA, Buist AS, Calverley PM, Jenkins CR, Hurd SS; GOLD Scientific Committee. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease. NHLBI/WHO Global Initiative for Chronic Obstructive Lung Disease (GOLD) Workshop summary. *Am J Respir Crit Care Med* 2001; **163:** 1256-1276.
21. American Thoracic Society. Evaluation of impairment/disability secondary to respiratory disorders. *Am Rev Respir Dis* 1986; **133:** 1205-1209.
22. American Thoracic Society. Guidelines for the evaluation of impairment/disability in patients with asthma. *Am Rev Respir Dis* 1993; **147:** 1056-1061.

Notes