



# Malta Guidelines for the Management of Chronic Obstructive Pulmonary Disease

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# introduction

Chronic Obstructive Pulmonary Disease (COPD) is an important cause of ill-health and death in Malta, especially among men. Currently available data concerning smoking patterns, predicts that prevalence of COPD will continue rising principally because of the increasing incidence of smoking among women. Indeed some studies suggest that women are more susceptible to the effects of tobacco smoke than men.

At St Luke's Hospital, every day of the year sees patients admitted for treatment of exacerbations of the disease: this group of patients is only a part of the severe portion of the spectrum of COPD. At a global level, COPD ranks as the 4<sup>th</sup> leading cause of death, and whereas cerebrovascular and heart disease rates are gradually declining, COPD rates are on the increase.

The total burden of COPD is greatly underestimated, as the disease is often not diagnosed until it is moderately advanced. In the early stages of the disease, it may only present during winter-time as a complication of a common cold. Apart from the negative impact on the health and quality of life of individuals, the socioeconomic effect of COPD involves both direct and indirect health-care costs. The direct costs are usually progressive and include not only hospital inpatient and outpatient care but also visits to family doctors, domiciliary care and support services. The progressive nature of COPD often forces patients into early retirement and this is usually preceded by an increasing number of days off work, for both patient and carer. An indirect cost to the patient and family is in the form of lost income related to disability and premature death.

The scope of these clinical practice guidelines is to increase awareness of COPD among health professionals and to help reduce morbidity and mortality from this often relentless condition. These guidelines are designed to provide direction for specific situations within the spectrum of COPD and they are not intended to override clinical judgement.

This document has been produced as part of the Global Initiative for Chronic Obstructive Lung Disease (GOLD) conducted in collaboration with the US National Heart, Lung and Blood Institute (NHLBI) and the World Health Organisation (WHO). GOLD aims to improve prevention and management of COPD through a concerted worldwide effort of people involved in all areas of healthcare and healthcare policy, and to encourage renewed research interest in this extremely prevalent disease.

The material presented is as far as possible based on published evidence and has been adapted for use in Malta taking into account the local health-care system and practice. In the main it was drawn from two major sources: the NHLBI/WHO Workshop Report *Global Strategy for the Diagnosis Management and Prevention of COPD*, and the European Respiratory Society Monograph *Management of Chronic Obstructive Pulmonary Disease*.

These guidelines were prepared by Prof. Joseph Cacciottolo and Dr. Maria Cordina. They were reviewed by, and discussed with a panel of experts and professional user groups in the health field. This process ensured that in addition to being evidence-based, these guidelines represent consensus among a core group of health-care professionals who are actively involved in clinical practice in Malta.

# what is COPD?

Chronic obstructive pulmonary disease is characterised by airflow limitation that is not fully reversible. The airflow limitation is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases.

The development and progression of COPD depends on the individual's particular susceptibility and there is evidence that genetic factors predispose to the disorder. Patients with COPD tend to deteriorate progressively especially if there is recurrent exposure to noxious agents. Once exposure is stopped, decline in lung function may decrease, and progression of the disease may be slowed or even arrested.

The symptoms of COPD, the functional abnormalities and the secondary complications can all be explained on the basis of this underlying inflammation and consequent damage to the airways and lung tissue.

The terms *chronic bronchitis* and *emphysema* were previously used to describe this disease, however they are not included in the present definition as neither describes COPD accurately. They are still useful terms: *chronic bronchitis* is a clinical term that is also useful for epidemiological studies. *Emphysema* is a pathological term characterised by destruction of alveoli.

### Pathophysiology

Repeated exposure to noxious particles and gases, such as the oxidant compounds in cigarette smoke, set off a series of pathological changes in the lungs that may result in COPD. The disorder is characterised by chronic inflammation throughout the airways, parenchyma and pulmonary vasculature. There is also impairment of the normal defensive and repair mechanisms of the lungs. Initially, the effects of these abnormalities only become apparent on exercise due to impaired mechanics of breathing. As the disease progresses, they also become manifest at rest.

Initial physiological changes include mucus hypersecretion, ciliary dysfunction, airflow limitation and pulmonary hyperinflation. As COPD advances, gas exchange abnormalities become apparent and the end stages of the disease are marked with pulmonary hypertension and subsequently cor pulmonale.

### Differences between COPD and Asthma

Although inflammation is the pathological basis of both COPD and asthma, the inflammatory response is different. Both diseases may co-exist, and inflammation in the lungs may then show both features of asthma and COPD. The inflammation in asthma is characterised by an increase in eosinophils and CD4<sup>+</sup> T lymphocytes, a small increase in macrophages and activation of mast cells. In COPD, the predominant cell type is the neutrophil with a large increase in macrophages and CD8<sup>+</sup> T lymphocytes.

A thorough clinical assessment, together with a chest X-ray (CXR) and spirometric evaluation is essential. A significant bronchodilator response (>12%) strongly suggests asthma. When asthma and COPD coexist, both present similar major symptoms, however these vary from day to day in asthma, while they are slowly progressive in COPD. A major distinguishing feature is that airflow limitation in asthma is to a large extent reversible, while in COPD it is mostly irreversible and progressive. As regards treatment, corticosteroids have limited use in COPD, while in asthma they have a central role in inhibiting inflammation.

# What causes COPD?

## what causes COPD?

### Risk Factors

The factors which predispose a person to developing COPD, are either host factors or environmental exposures and it is usually an interaction between these sets of factors that give rise to COPD. The nature and strength of interactions between risk factors provide a possible explanation as to why not all smokers with a similar smoking history go on to develop COPD.

## Host Factors

### Alpha-1 Antitrypsin Deficiency

Hereditary deficiency of alpha-1 antitrypsin is the best documented risk factor for COPD. Alpha-1 antitrypsin is the major inhibitor of elastase released by neutrophils during inflammation. This deficiency is rare and only explains a very small proportion of COPD cases, however it demonstrates the interaction between host factors and environmental factors. Premature and accelerated development of emphysema and decline in lung function occur in both smokers and non-smokers with severe deficiency, although smoking greatly increases the risk.

### Airway Hyperresponsiveness

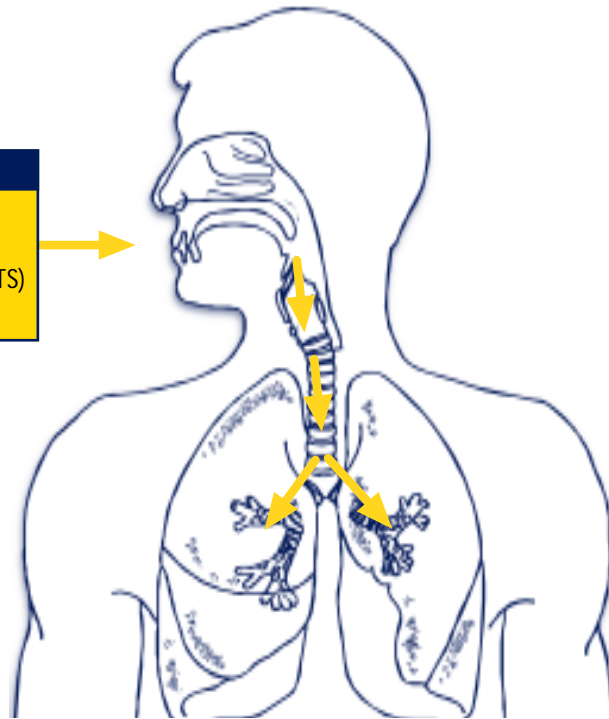
Persons with a history of asthma and airway hyperresponsiveness have an increased risk of developing COPD.

### Lung Growth

Reduced lung growth due to factors operating during gestation or exposure to noxious fumes such as cigarette smoke during childhood may predispose to the later development of COPD.

#### Inhaled Particles

Cigarette smoke  
Occupational Dusts & Chemicals  
Environmental Tobacco Smoke (ETS)  
Indoor/Outdoor Air Pollution



#### Host Factors

Genetic (eg. alpha-1 antitrypsin deficiency)  
Airway hyperresponsiveness  
Lung growth

#### Exposures

Tobacco smoke  
Occupational dusts and chemicals  
Indoor and outdoor air pollution  
Infections  
Socioeconomic status

## Exposures

### Smoking

Cigarette smoking is by far the most important risk factor for COPD. Smokers have a higher prevalence of respiratory symptoms and lung function abnormalities. They have a steeper rate of decline in FEV<sub>1</sub> and a higher death rate than non-smokers.

Differences in morbidity and mortality between cigarette smokers and non-smokers increase in direct proportion to the amount of smoking. Among individuals who smoke more than 25 cigarettes/day, the risk of death from COPD increases more than twenty-fold. Passive smoking may also increase risk of developing COPD by increasing the total burden of inhaled particles and gases. Smoking during pregnancy may affect foetal lung development and may possibly prime the immune system.

### Occupational Dusts and Chemicals

Increased risk of COPD has been described in relation to several occupations involving exposure to dusts, fumes and solvents. This risk is increased when there is concurrent cigarette smoking.

### Indoor and Outdoor Air Pollution

Air pollution is detrimental to persons with lung disease. However, the precise manner in which this factor contributes to the development of COPD is still unclear.

**Infections**

A history of severe infections, especially those of viral nature, during childhood is associated with reduced lung function and may be a contributing factor to the development of COPD in later life.

**Socioeconomic Status**

Evidence suggests that persons having a lower socioeconomic status are at a higher risk of developing COPD. This may also be because of a combination of such factors as higher smoking prevalence, poor nutritional status and type of occupation.



# s COPD present?

## how does COPD present?

**The Symptoms**

Patients with COPD typically present during the 4<sup>th</sup> to 5<sup>th</sup> decade. They usually seek medical help because of *breathlessness*, initially on exertion. The other common symptom of COPD is *cough*, which is often accompanied by sputum production. When the disease is mild or in its initial stages patients are practically asymptomatic; they only complain of what they call a 'smokers' cough' and shortness of breath when their physical capacity is stretched.

Typically there is also a history of long-term and/or heavy smoking. Indeed, if a history of smoking is absent and the patient is relatively young, there should be screening for asthma and alpha-1 anti-trypsin deficiency.

## Cough

In most patients with COPD, cough either precedes onset of breathlessness or appears concurrently with it. It is often dismissed by the patient as an expected consequence of smoking or environmental exposure. Cough in COPD is typically worse in the morning and usually appears within 10 years of the start of regular smoking. Later, this symptom is present daily and often throughout the day. Daily cough, productive of sputum for three consecutive months for at least 2 years running, is a useful clinical definition of chronic bronchitis.

During exacerbations of COPD, sputum increases in volume and becomes purulent. Sputum production is however difficult to evaluate as patients may swallow the sputum rather than expectorate it.

Coughing diminishes or disappears in a large number of patients who stop smoking, however lung function abnormalities persist.

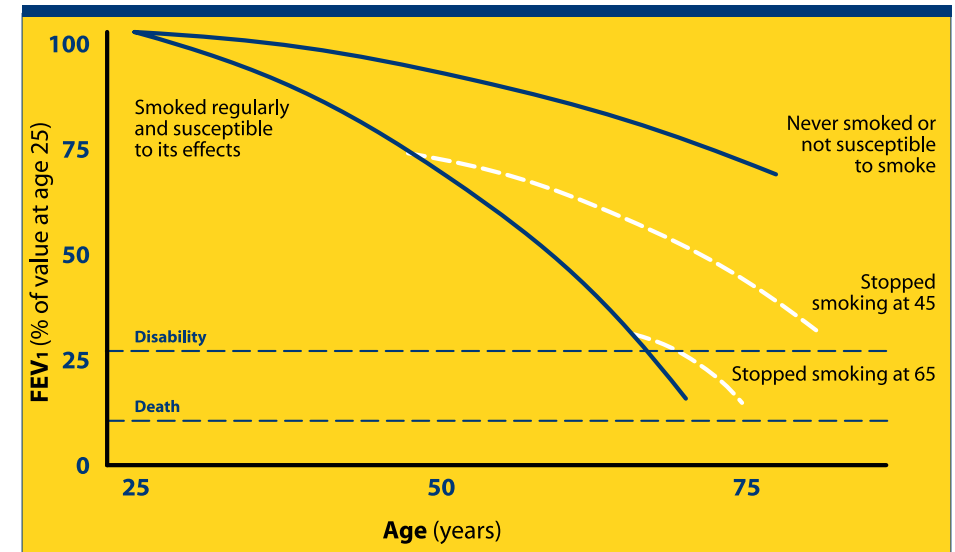
## Breathlessness

Shortness of breath is the reason most patients seek medical attention and it is a major cause of disability and anxiety. The symptom is persistent and often progressive. Patients with COPD experience breathlessness at lower levels of exercise than unaffected people of the same age and is initially only noted on unaccustomed effort.

Onset of breathlessness or awareness of difficulty with breathing is very gradual and behaviour can be modified to avoid its onset or limit its effect. Later on, further adaptation occurs and patients may alter their breathing pattern to minimise the sensation of shortness of breath.

As lung function deteriorates, breathlessness becomes more hindering and often conditions everyday activities. When extra efforts are required, patients rely increasingly on accessory muscle groups to support ventilation. Eventually breathlessness disables the patient and interferes with such essential activities as washing and dressing. It severely impairs health-related quality of life and confines the patient to the home.

Decline in forced expiratory volume in one second FEV<sub>1</sub> (presented as a percentage of the value at 25 years of age). Natural decline with age and accelerated decline in susceptible smokers. Rate of decline in individuals who stop smoking may decrease to values observed in those who never smoked.



Fletcher C and Peto R (1977)

## Other Symptoms

### Wheezing

This symptom may vary and because of its often intermittent nature, it is not easy to evaluate.

### Chest Tightness

This often follows exertion, is poorly localised and is 'muscular' in character. It may be difficult to distinguish between the pain of ischaemic heart disease or gastroesophageal reflux disease, both common problems that might co-exist among middle aged smokers.

### Haemoptysis

This symptom is often a source of concern as smoking is a powerful risk factor for both COPD and lung cancer. It should always be investigated, and bronchoscopic evaluation is often necessary.

### Anorexia and Weight Loss

Both are common problems as COPD advances and weight loss is associated with a worsening prognosis.

## The Signs

Physical signs caused by airflow limitation are usually absent or subtle until significant functional impairment has occurred. Signs become quite evident and specific in advanced COPD.

- ▲ Chest wall abnormalities are the result of over-inflation of the lungs. The chest becomes 'barrel-shaped' and the abdomen protrudes.
- ▲ The respiratory rate is often increased and breathing is shallow. Patients commonly purse their lips on exhaling in order to slow and control expiratory flow.
- ▲ The accessory respiratory muscles (sternocleidomastoid and scalene) are used when the disease is advanced.
- ▲ Palpation and percussion are usually unhelpful in COPD. Cardiac dullness is reduced, the cardiac apex beat may be difficult to locate and the liver is often displaced downwards.
- ▲ The breath sounds are reduced and the expiratory phase often prolonged and accompanied by wheezing, although the latter feature is not specific to COPD.
- ▲ When pulmonary hypertension and right ventricular failure complicate COPD, the clinical signs are distended neck veins, liver enlargement and dependent oedema.
- ▲ Ankle swelling may result from cor pulmonale, often in combination with immobility forced on the patient by decreased exercise tolerance.
- ▲ Cyanosis and coarse tremor often occur in advanced COPD and reflect the severe hypoxaemia and hypercapnia.

# COPD assessed?

how is COPD assessed?

Assessment of patients with COPD is principally based on the clinical evaluation and lung function tests. Although the physical examination is an important part of patient care, it is rarely diagnostic in COPD. Physical signs of airflow limitation are seldom present until significant impairment of lung function has occurred, and their detection has a relatively low sensitivity and specificity.

### Spirometry

A diagnosis of COPD suggested by the presenting signs and symptoms is confirmed by spirometry, as this is a standardized and objective way of measuring airflow limitation. Spirometry measures the forced vital capacity (FVC) and the forced expiratory volume in 1 second (FEV<sub>1</sub>). The ratio of these measurements FEV<sub>1</sub>/FVC is then calculated.

A finding of an FEV<sub>1</sub> of less than 80% of the predicted value after bronchodilatation and an FEV<sub>1</sub>/FVC below 70% confirms the presence of airflow limitation that is not fully reversible.

Spirometry is essential for the accurate diagnosis and periodic assessment of COPD. Although peak expiratory flow (PEF) is sometimes used as a measure of airflow limitation, it may underestimate the degree of obstruction, and a low flow rate is not specific to COPD.

Among persons at risk for COPD, symptoms may be minor and occasional, while spirometry may only be slightly abnormal. From a preventive public health aspect these individuals should be identified and convinced that minor respiratory symptoms may be the markers of future ill-health.

COPD is usually a progressive disease and lung function can be expected to worsen over time, even with the best available care. As COPD increases in severity, visits to the family doctor, the health centre and the hospital increase in frequency. Regular assessment should include evaluation of:

- ▲ Exposure to risk factors, especially active *and* passive smoking
- ▲ Treatment regimens and compliance to advice
- ▲ Exacerbations of the disease
- ▲ Development of complications

### Complete Blood Count

A haematological complication of COPD is polycythaemia and this in turn may predispose to vascular complications. It should be suspected, when the haematocrit is over 47% in females and over 52% in males (or haemoglobin levels of over 16g/dl in females and 18g/dl in males).

### Radiology and Echocardiography

There are no specific features of COPD on a plain CXR and the ones that are described are those of hyperinflation resulting from emphysema (flattened diaphragm, increase in the retrosternal airspace, hyperlucency of the lung fields and rapid tapering of the vascular markings). Right ventricular hypertrophy produces non-

Besides lung function tests, other tests may be used to assess COPD and its complications.

specific cardiac enlargement on the plain CXR, while pulmonary hypertension causes an increase in the width of the descending right pulmonary artery measured just below the hilum. However, a better non-invasive assessment of pulmonary arterial pressure is a Doppler echo test.

### Classification of COPD by Severity

#### Stage 0: At risk

Symptoms: Chronic cough and sputum production: often ignored or disregarded by patients as insignificant.

Spirometry: Normal.

#### Stage I: Mild COPD

Symptoms: May be asymptomatic, but there is usually chronic cough and sputum production.

Spirometry: FEV<sub>1</sub>/FVC less than 70%.  
FEV<sub>1</sub> above 80% of the predicted value.

#### Stage II: Moderate COPD

Symptoms: Breathlessness often interfering with daily activities. May be asymptomatic, but there is usually chronic cough and sputum production.

Spirometry: FEV<sub>1</sub>/FVC less than 70%.  
FEV<sub>1</sub> below 80% of predicted value (IIA).  
FEV<sub>1</sub> below 50% of predicted value (IIB).  
(exacerbations more frequent in this group).

#### Stage III: Severe COPD

Symptoms: Worsening cough, sputum production and severe breathlessness.

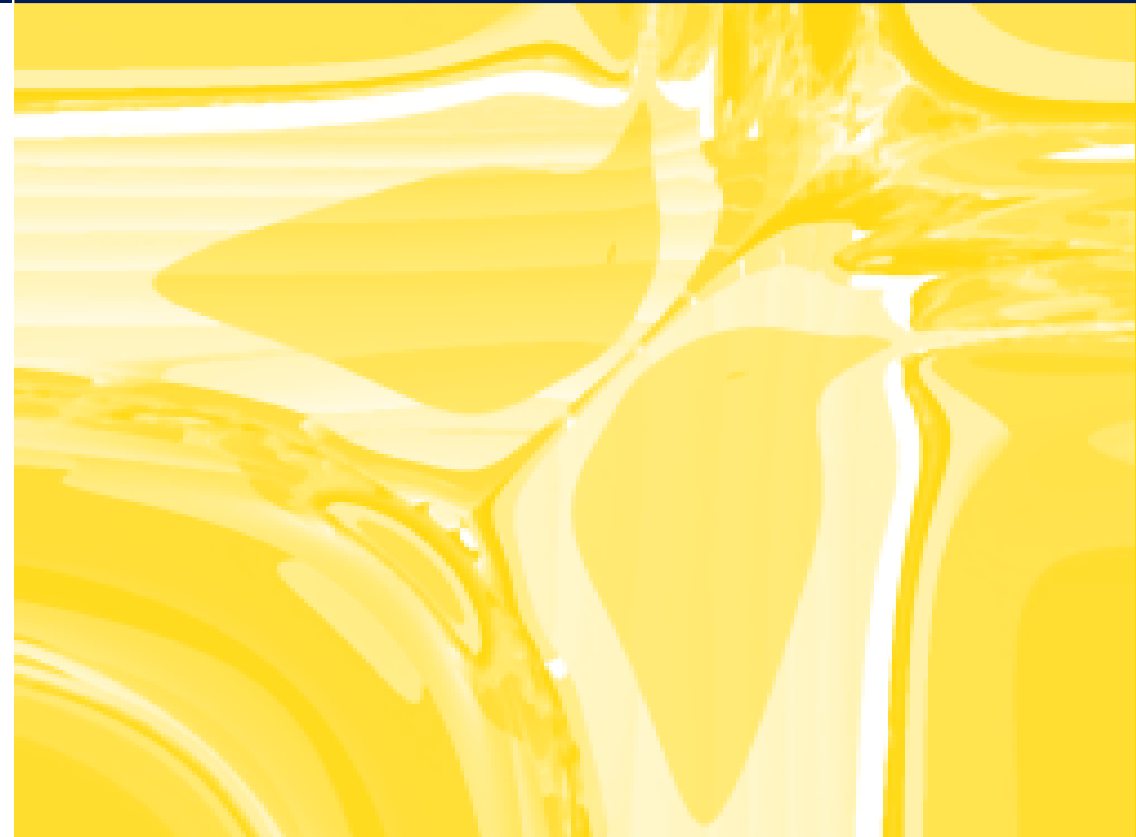
Spirometry: FEV<sub>1</sub>/FVC less than 70%.  
FEV<sub>1</sub> below 30% of predicted value or  
FEV<sub>1</sub> below 50% together with respiratory failure or clinical signs of right heart failure.

At Stage III patients are markedly disabled and exacerbations may be life threatening.

In the assessment of patients with moderate or severe stable COPD, measurement of arterial blood gases, with the patient breathing room air, is recommended.

Other than in pathophysiological terms, COPD may be assessed in terms of impact of the disease on patients' health-related quality of life: indeed this is often what matters most to the individual.

Limitation of exercise tolerance and restriction of functional independence are important factors leading to a decline in the quality of life. Difficulty with breathing during exercise and at rest is often a cause for anxiety and leads to patients' sense of loss of control over their disease and their lifestyle. Chronic cough, sputum production, and nocturnal hypoxaemia may cause sleep disturbance and raise specific social and emotional problems in affected patients and their families.



# COPD managed?

## how is COPD managed?

Prevention of COPD is the ultimate goal. Once the disease has been diagnosed, effective management depends on an assessment of the severity of the disease. Management of COPD requires a multi-disciplinary team approach often involving the family physician, the respiratory physician, nursing staff, pharmacists and physiotherapists. The support of close family and friends is often crucial, especially when COPD is severe, exacerbations frequent and disability marked.

**The goals of management are to:**

- ▲ relieve symptoms and improve quality of life
- ▲ slow down the decline in lung function and disease progression
- ▲ prevent and effectively treat exacerbations
- ▲ prevent and treat complications

The goals of management should be reached with minimal side-effects from treatment, a particular challenge in COPD patients as they often have concurrent chronic disorders.

In order to prevent or slow down disease progression, it is necessary to reduce decline in lung function. Serial measurements of FEV<sub>1</sub> are essential to monitor disease progression, and a fall of more than 50ml/l per year implies accelerated decline.

**Cessation of Smoking**

As cigarette smoking is the major cause of development of COPD, cessation of smoking is an essential component at all stages of management of the disease. Patient education regarding smoking cessation has the greatest capacity to influence the natural history of the disease. When smokers quit their habit, the annual rate of decline in lung function slows down and approaches that of non-smokers.

Each contact between a health professional and any patient who smokes should be used to reinforce advice about stopping smoking. In this context, every effort should be made not only to stop children from taking up the smoking habit but also from being passively exposed to tobacco smoke. Health-care professionals also have an obligation to support the general public health measures against smoking. This starts with not smoking themselves and leads on to support for changes in legislation to curb tobacco advertising and sponsorship.

**How important is smoking cessation in the management of COPD?**

- ▲ Smoking cessation is the single most effective way to arrest the progression of COPD.
- ▲ Effective smoking cessation initiatives are multifactorial and doctors, nurses, pharmacists, physiotherapists, and other health-care professionals have a key role in ensuring successful outcomes.
- ▲ Tobacco dependence is a chronic disease and for most people results in true drug dependence comparable to those caused by heroin and cocaine.
- ▲ Tobacco users should be identified by health-care professionals and strongly advised to stop at every available opportunity. The message is reinforced by linking smoking to the signs and symptoms of their disease.
- ▲ Having assessed the users' willingness to stop smoking, the patient should be assisted to stop by offering counselling together with a personalised plan. Provision of social support during the treatment period is important and failure to provide support may easily lead to relapse.
- ▲ Pharmacotherapy, such as nicotine replacement and bupropion, should be appropriately prescribed in the absence of any contraindications.
- ▲ Health care professionals should maintain contact with participants in smoking cessation programmes and follow them up on a regular basis.
- ▲ It is accepted that the possibility of relapse is high, as this is in the nature of dependence and not the failure of the patient or the clinician. An understanding and supporting attitude often encourages the smoker to attempt to stop once again.

## Therapeutic management

The overall approach to managing stable COPD should be characterised by a stepwise increase in treatment depending on the severity of disease.

### Bronchodilators

Bronchodilator medications are central to the symptomatic management of COPD; they may either be given on a regular basis to prevent and control symptoms or on as-needed basis for relief of symptoms. Bronchodilators are best given by inhalation in order to have a fast onset of action and to reduce potential side-effects.

Inhaled bronchodilators fall into two groups,  $\beta_2$  agonists and anticholinergics. The bronchodilator effects of short-acting  $\beta_2$  agonists (e.g. salbutamol and terbutaline) last about 4 hours while long-acting  $\beta_2$  agonists (salmeterol and formoterol) last about 12 hours, with formoterol showing a relatively faster onset of bronchodilator effect. The chief adverse effects of  $\beta_2$  agonist bronchodilators are resting sinus tachycardia and tremor, especially in elderly patients.

The bronchodilator effects of the short-acting anticholinergic agent ipratropium lasts about 6 hours while the effects of tiotropium last about 24 hours. As anticholinergic drugs are poorly absorbed, their incidence of side-effects is low and they are safe in a wide range of doses in clinical settings.

To achieve the most effective regimen with least side effects, treatment should start with a single inhaled drug; however combining  $\beta_2$  agonist and anticholinergic bronchodilators may improve efficacy and decrease the risk of side-effects, compared to increasing the dose of a single bronchodilator.

If despite using a combination of  $\beta_2$  agonists and anticholinergic bronchodilators, respiratory symptoms are troublesome, an oral slow-release methylxanthine preparation e.g. theophylline should be considered. Use of methylxanthines is often limited by their side-effects which include nausea, vomiting, arrhythmias, convulsions and interactions with other drugs, and their narrow therapeutic window.

### Corticosteroids

Whereas corticosteroids are central to treatment among those suffering from asthma, they are of limited use among patients with COPD. The role of corticosteroids in COPD is limited to specific indications such as a short course of intravenous or oral treatment during exacerbations, or if there is specific symptom and lung function response. Inhaled corticosteroids do not slow the decline in FEV<sub>1</sub>, and have a minimal clinical effect in patients with COPD. In patients with advanced disease they have, however, been shown to reduce the frequency of exacerbations.

### Mucolytic Agents

A few patients having viscous sputum may find mucolytic agents useful, however the overall benefits are small and their widespread use is not supported by scientific evidence.

### Oxygen

Oxygen therapy is a principal component of treatment for patients with severe COPD and the aim of therapy is to improve the PaO<sub>2</sub> to >60mmHg (8kPa) or an SaO<sub>2</sub> of >90%. The primary haemodynamic effect of oxygen therapy is to prevent progression of pulmonary hypertension.

Oxygen may be used occasionally to relieve breathlessness or to increase tolerance to exercise. Long-term oxygen treatment (over 15 hours/day) is the one treatment apart from smoking cessation that has been shown to increase life-expectancy in COPD. It is crucial to determine hypoxaemia over a stable period of 2-4 weeks with optimal treatment before prescribing long-term oxygen therapy.

It is neither practical nor economical to provide long-term oxygen therapy using compressed gas cylinders. When there are clear clinical indications for long-term oxygen needs, domiciliary oxygen concentrators are the safest and most practical means of delivery.

### Pulmonary Rehabilitation

A programme of pulmonary rehabilitation has been shown to improve health-related quality of life and reduces the need for hospitalisation. It often reduces anxiety and depression associated with COPD. Motivated patients benefit from rehabilitation programmes involving a multi-disciplinary team and addressing exercise training, nutritional advice and patient education.

### Therapy at Each Stage of COPD

All patients should be advised about avoidance of risk factors and be vaccinated against influenza and pneumococcal infections. Treatment for COPD and other conditions, should be regularly monitored, taking care to avoid medications which interact with current treatment regimens and those which could provoke an exacerbation.

#### Stage I: **Mild COPD**

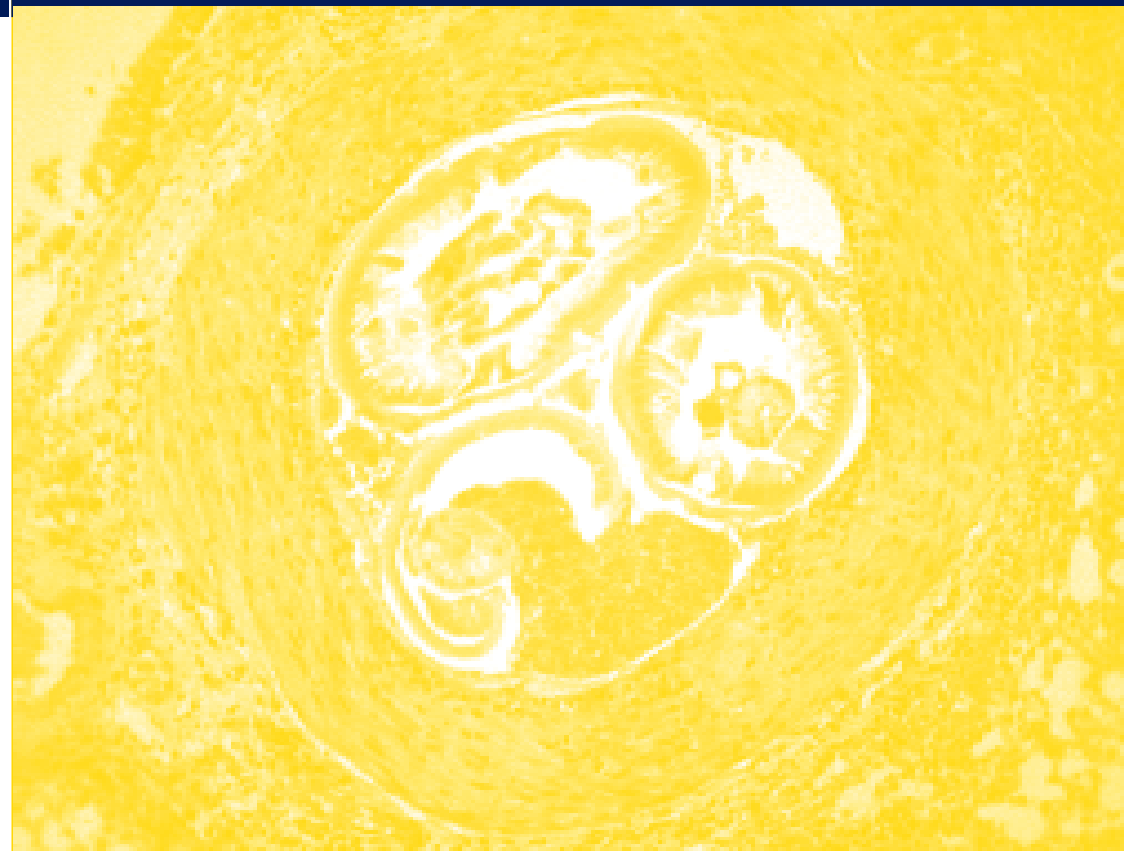
- ▲ Short-acting bronchodilator when needed

#### Stage II: **Moderate COPD**

- ▲ Rehabilitation
- ▲ Regular treatment with one or more bronchodilators
- ▲ Inhaled corticosteroids if there is a significant symptomatic and lung function response or if there are repeated exacerbations

#### Stage III: **Severe COPD**

- ▲ Regular treatment with one or more bronchodilators
- ▲ Inhaled corticosteroids if there is a significant symptomatic and lung function response or if there are repeated exacerbations
- ▲ Treatment of complications
- ▲ Rehabilitation
- ▲ Long-term oxygen therapy if there is respiratory failure



# Exacerbations managed?

When COPD increases in severity, there are often episodes of acute exacerbations of symptoms and the need for medical attention becomes necessary as airflow limitation worsens. On average, patients have one to four acute exacerbations per year and hospitalisation is often needed to manage a severe event.

An exacerbation is an acute event superimposed on stable COPD. There is usually *increased breathlessness* which may be accompanied by wheezing and chest tightness. Coughing increases and there are *changes in both volume and colour of sputum*. Exacerbations may be accompanied by fever, fatigue, sleep disturbance, depression and mental confusion.

Acute exacerbations are most often caused by an infection in the tracheobronchial tree, frequently viral in nature. Air pollution is another common cause of exacerbation. In about one-third of severe exacerbations a cause cannot be identified.

#### Common causes of acute exacerbation of COPD

- |                   |  |
|-------------------|--|
| <b>Primary:</b>   | <ul style="list-style-type: none"> <li>▲ tracheobronchial infection</li> <li>▲ air pollution</li> </ul>  |
| <b>Secondary:</b> | <ul style="list-style-type: none"> <li>▲ pneumonia</li> <li>▲ pulmonary embolism</li> <li>▲ pneumothorax</li> <li>▲ inappropriate use of sedatives, narcotics and beta blocking agents</li> <li>▲ inappropriate use of oxygen</li> <li>▲ right and/or left heart failure or arrhythmias</li> </ul> |

Information on the history of the previous stable condition is essential as this gives insight into the usual level of activity of the patient before the exacerbation. The duration and progression of symptoms should be determined and the regular and more recently taken medications should be reviewed. Among the indices of severity, the doctor should consider worsening or new-onset central cyanosis, development of dependent oedema and haemodynamic instability. The most important sign of a severe exacerbation is a change in the alertness of the patient and this in itself indicates an urgent need for hospitalisation.

## Home Management of an Acute Exacerbation

When an exacerbation is not deemed severe and in situations where there is good social support and supervised care, home management of an exacerbation is appropriate.

#### The objectives of home management are to:

- ▲ treat infection if present
- ▲ remove excess bronchial secretions
- ▲ maximise airflow
- ▲ improve respiratory muscle strength and thus to facilitate cough
- ▲ avoid or monitor adverse effects of treatment
- ▲ educate patient and family on signs of deterioration and action to be taken

As a first step initiate or increase the dose of inhaled bronchodilator therapy. If not already used, add inhaled ipratropium. Consider prescribing an antibiotic; these are however only effective when there is also increased sputum volume or purulence. In this case consider an oral broad spectrum antibiotic such as amoxicillin/clavulanic acid or an extended-spectrum macrolide, or co-trimoxazole. Whenever possible, short sharp courses of antibiotics should be given. If the need for multiple courses arises, a rotational system may be used to minimise the emergence of resistant strains of organisms. Encourage fluid intake and sputum clearance by coughing and avoid sedative or hypnotic medication. Initiation of oxygen therapy at home during an exacerbation of COPD may lead to serious complications and must be avoided. The decision to initiate oxygen therapy should be taken in consultation with a respiratory physician.

If despite optimum home care, there is lack of response to initial medical management within 48 hours or there is an increase in the intensity of symptoms such as sudden onset of resting dyspnoea, the patient should be hospitalised.

## Hospital Management

### The objectives of hospital management of COPD are to:

- ▲ properly evaluate the severity
- ▲ identify life-threatening episodes
- ▲ identify the cause(s) of the exacerbation and treat accordingly
- ▲ administer controlled oxygen therapy
- ▲ provide assisted ventilation if indicated
- ▲ return patients to their best previous condition

On admission to the A+E Department the patient with an exacerbation of COPD should be given controlled oxygen. Start therapy with low concentrations of oxygen using a Venturi mask delivering 24% or nasal cannulae with a flow rate of 1-2 litres per minute. Monitor blood gases and/or oxygen saturation (SaO<sub>2</sub>), with the goal of treatment being to raise PaO<sub>2</sub> to 60mmHg (8kPa) or SaO<sub>2</sub> to >90%.

It must be rapidly determined whether the condition is life-threatening, and therefore requiring admission to the ITU for assisted ventilation. Use of ventilation in end-stage COPD patients is influenced principally by the likely reversibility of the precipitating event and the patient's wishes. Major complications include the risk of ventilator-acquired pneumonia, especially when multi-resistant organisms are present, and failure to wean to spontaneous breathing.

Non-invasive mechanical ventilation is a means of avoiding many of the complications associated with invasive ventilatory supports, and should be attempted whenever possible. Ideally, the decision to ventilate patients with an exacerbation complicating severe and long-standing COPD should be taken in consultation with their own respiratory physician.

### Bronchodilators

Increased doses and/or combinations of salbutamol plus ipratropium should be administered by nebulisation preferably driven by compressed air rather than oxygen. During nebulisation, if supplementary oxygen is needed, it should be given by nasal cannulae.

### Corticosteroids

Oral or intravenous corticosteroids should be administered in addition to oxygen and bronchodilator therapy. A 10 day course of prednisolone, 30mg daily, is usually beneficial. Prolonged treatment does not result in greater efficacy and may result in acute side effects such as psychiatric disturbance and hyperglycaemic episodes.

### Antibiotics

A combination of different types of antibiotics is sometimes required and if there is evidence of severe infection, they should be initially administered intravenously. Sputum cultures are recommended in hospitalised patients, however as sensitivity results are only available after a few days, empirical treatment with antibiotics of the cephalosporin and the macrolide classes is usually started on admission. The newer fluoroquinolones are also useful in such situations.

### Other Measures

These include physiotherapy, supplementary nutrition and treatment with subcutaneous heparin, when patients are immobilised, polycythaemic or dehydrated. Mobility in bed should be encouraged early on as a precursor to ambulation.

## When is the patient ready for discharge?

Patients with COPD are ready for discharge to their normal environment and to the care of their family doctor when inhaled bronchodilator therapy is not required more frequently than every 4 hours.

If previously ambulatory, patients should be able to walk across a room, and eat and sleep without frequent awakening due to breathlessness. The clinical status and blood gases should be stable for 24 hours before discharge and the patient, family and physician should be confident of the ability to cope in a home environment.

A detailed report should invariably be forwarded to the patient's family doctor. This ensures continuity of care; an essential feature in the management of patients with COPD.

30 Signs and Symptoms	Spirometry	Pharmacological Treatment in Stable COPD	Non-Pharmacological Treatment	Patient Education	31
<b>All Stages</b>					
<ul style="list-style-type: none"> <li>▲ Influenza vaccine</li> <li>▲ Address the risk posed by smoking and other factors. Encourage smoking cessation.</li> </ul>					
<b>Stage 0: At Risk</b>					
Chronic cough Sputum production	▲ Normal	▲ Pneumococcal vaccine		▲ Identify individual risk factors and advise on avoidance strategies.	
<b>Stage I: Mild</b>					
Usually with chronic cough and sputum production	▲ Mild airflow limitation FEV <sub>1</sub> /FVC <70% FEV <sub>1</sub> ≥80% predicted	▲ Short acting bronchodilator prn: Inhaled salbutamol 100-200µg or terbutaline 250-500µg		<ul style="list-style-type: none"> <li>▲ Explain the nature of COPD.</li> <li>▲ Explain the prescribed therapy.</li> <li>▲ Instruct on inhaler technique, spacer and nebulizer use.</li> <li>▲ Inform how to recognise and treat acute exacerbations.</li> <li>▲ Outline strategies for reducing shortness of breath.</li> </ul>	
<b>Stage II: Moderate</b>					
Usually with chronic cough and sputum production and breathlessness interfering with daily activities	▲ Worsening airflow limitation FEV <sub>1</sub> /FVC <70% 30% ≤ FEV <sub>1</sub> <80% predicted IIA: 50% ≤ FEV <sub>1</sub> <80% IIB: 30% ≤ FEV <sub>1</sub> <50%	<ul style="list-style-type: none"> <li>▲ Regular treatment with one or more bronchodilators: Inhaled salbutamol 100-200µg or terbutaline 250-500µg. Formoterol 12-24µg or salmeterol 50-100µg and ipatropium 40-80µg or tiotropium 18µg.</li> <li>▲ Inhaled corticosteroids only if patient's symptoms and lung function improve or if patient has repeated exacerbations.</li> </ul>	▲ Rehabilitation	▲ As above	
<b>Stage III: Severe</b>					
Worsening cough, sputum production, severe breathlessness	▲ Severe airflow limitation FEV <sub>1</sub> /FVC <70% FEV <sub>1</sub> <30% predicted or FEV <sub>1</sub> <50% plus respiratory failure or cor pulmonale	<ul style="list-style-type: none"> <li>▲ As above</li> <li>▲ Treat complications</li> </ul>	<ul style="list-style-type: none"> <li>▲ Rehabilitation</li> <li>▲ Oxygen therapy on a long-term basis.</li> </ul>	<ul style="list-style-type: none"> <li>▲ Explain about the possibility of complications.</li> <li>▲ Instruct on the use of long term oxygen treatment and the use of oxygen concentrators.</li> </ul>	

FEV<sub>1</sub>: Forced Expiratory Volume in one second  
FVC: Forced Vital Capacity

Respiratory Failure: PaO<sub>2</sub> <8.0kPa (60mm Hg) with or without PaCO<sub>2</sub> >6.7kPa (50 mm Hg) while breathing room air.

Doses: β<sub>2</sub> agonists refer to average dose given up to 4 times daily for short-acting and 2 times daily for long-acting preparations. Short-acting anticholinergics are usually given 3-4 times daily and the long-acting anticholinergic, once daily.

