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An Roinn Coimirce Sóisialaí
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Department of Social Protection

Chronic Obstructive Pulmonary Disease

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1.1 Overview of Chronic Obstructive Pulmonary Disease

Chronic Obstructive Pulmonary Disease (COPD) is a chronic, progressive disorder characterised by reduced airflow on expiration due to airway obstruction that is not fully reversible and usually worsens over time (GOLD - Global Initiative for Chronic Obstructive Lung Disease, 2009). The condition may be accompanied by airway hyper-reactivity, and can be associated with an abnormal reaction to inhaled particles or gasses. COPD is not a single condition, but is an 'umbrella' term covering a number of different types of the disorder, including the more familiar terms of chronic bronchitis and emphysema which are now described as COPD (WHO, 2009).

COPD is a preventable condition (WHO, 2009); the primary cause is exposure to tobacco smoke (including passive smoking). Other causes of the condition include exposure to indoor or outdoor pollution (for example, exposure to smoke from wood-burning stoves (which is increasing the prevalence in women in low income countries) and occupational exposure.

As a disorder, COPD is under-recognised and under-treated (World Health Organisation, 2009, GOLD, 2009). Although in the past, there was little that could be done to relieve COPD, in the past decade there has been significant research into the condition, leading to a number of new treatment options (NCGC – National Clinical Guideline Centre, 2009), including pharmacologic therapies such as long-acting bronchodilator drugs, and the development of respiratory rehabilitation services. Although the condition is not curable, the symptoms of the disease can be controlled with effective treatment.

The global burden of COPD is considerable. The disease is the fastest growing cause of death in the developed world (Murray and Lopez, 1996), and it is estimated that by 2030 COPD will move from its present position of the fourth leading cause of death to become the third leading cause of death and the fifth leading cause of disability in the world (World Health Organisation, 2009). Most of the epidemiological research has centred on high income countries, but it is thought worldwide that many of the more severe cases and deaths occur in low to middle income countries, with 90% of deaths from COPD estimated to occur in the latter group. Worldwide approximately 210 million people have COPD; with 80 million of these having moderate to severe forms of the condition. This resulted in 3 million deaths from COPD in 2005, approximately 5% of all deaths globally (WHO, 2009), and more than one fifth of all deaths due to respiratory disease (23%) (British Thoracic Society, 2006).

In economic terms, COPD is costly with direct healthcare costs across the European Union exceeding €38 billion (GOLD, 2009). In the UK, the direct healthcare costs result in COPD being one of the most costly conditions to treat for the National Health Service, with estimates of costs reaching around £800 million per year (Department of Health, 2005). Hospital inpatient costs of treating individuals with

COPD as inpatients come to half the amount that the NHS spends on disease annually (Healthcare Commission, 2006), and 1 in every 8 accident and emergency admissions in the UK is for COPD.

This economic impact is furthered by the fact that COPD has an extremely high occupational impact. It is estimated in the UK that the condition results in approximately 24 million lost work days annually (Anon, 2004, Department of Health, 2005; Healthcare Commission, 2006, NCGC, 2009). It has been estimated that approximately 40% of UK COPD suffers are below retirement age, with around one quarter of these unable to work due to their condition, and 10% limited in their ability to work (Britton, 2003)

DRAFT NOTE – National Institute of Clinical Excellence references throughout this document (referenced as NCGC - National Clinical Guideline Centre, 2009) have been taken from the *DRAFT* 2010 version of the chronic obstructive pulmonary disease: full guideline which is currently available for *CONSULTATION* at www.nice.org. DSP to confirm this is acceptable – previous guidelines are dated 2004. It is hoped the final version of this NICE guideline will be published whilst this protocol is in the DSP review process.

1.2 Definition of Chronic Obstructive Pulmonary Disease

Chronic Obstructive Pulmonary Disease is defined by the Global Initiative for Chronic Obstructive Lung Disease (GOLD) as:

'A preventable and treatable disease with some significant extrapulmonary effects that may contribute to the severity in individual patients. Its pulmonary component is characterised by airflow limitation that is not fully reversible. The airflow limitation is usually both progressive and associated with an abnormal response of the lungs to noxious particles or gases' (GOLD, 2009).

Chronic Obstructive Pulmonary Disease has been previously described by a number of other terms including Chronic Obstructive Airway Disease, chronic asthma, chronic bronchitis (chronic cough or mucus production for at least 3 months in at least 2 successive years when other causes of chronic cough have been excluded) and emphysema (abnormal permanent enlargement and damage of the air spaces distal to the terminal bronchioles).However these terms are no longer in general use but considered to be sub-conditions of a spectrum of disorders now generally termed as COPD (WHO, 2009, NCGC, 2009). COPD is differentiated from asthma in that the airway obstruction involved is not fully reversible, as with asthma (GOLD, 2009).

The symptoms of COPD include dyspnoea, a chronic cough and sputum production. A history of exposure to a risk factor (usually tobacco smoke) is almost always present but COPD can also occur in individuals who have not been exposed to a toxic or noxious substance – in these cases a genetic factor can be involved (see *Section 2 -Aetiology).* The symptoms of cough and sputum production may appear many years before any symptoms of airway obstruction occur – one of the reasons why the condition is under-recognised until advanced stages of the disease.

COPD additionally has effects on an individual which are not limited to pulmonary

function only. Weight loss, nutritional issues and musculo-skeletal issues are common effects of the condition, and evidence suggests that the existence of COPD will raise an individual's risk for developing other conditions including cardiac conditions such as myocardial infarction and angina, diabetes, lung cancer and respiratory infections (GOLD, 2009). However, because COPD often occurs in individuals who have been long terms smokers, it is also not unexpected that many such individuals also have other comorbid conditions which have arisen either due to smoking, or due to the process of aging (GOLD, 2009)

COPD can become exacerbated leading to an increase in the severity of symptoms and increasing functional impairment. There is no agreed clinical definition of a COPD exacerbation (NHS Institute for Innovation and Improvement, 2009).

COPD is disabling because of the reduced exercise tolerance resulting from impaired exchange of oxygen and carbon dioxide between the atmosphere and the pulmonary circulation.

1.3 International Classification of Diseases; 10th Edition (ICD-10) Classification

The World Health Organisation, in the 10th Edition of the International Classification of Diseases (ICD-10) (World Health Organisation, 2007); applies the following diagnostic classification for COPD:

J44: Other chronic obstructive pulmonary disease – includes chronic, bronchitis – asthmatic (obstructive), emphysematous but excludes asthma and Emphysema (see note below)

J44.0 Chronic obstructive pulmonary disease with acute lower respiratory infection

J44.1 Chronic obstructive pulmonary disease with acute exacerbation, unspecified

J44.8 Other specified chronic obstructive pulmonary disease

J44.9 Chronic obstructive pulmonary disease, unspecified

Although the ICD-10 classification for COPD excludes Emphysema as part of the diagnostic criteria, many research studies include the J43 group of diagnostic codes in addition to the J44 set for diagnostic purposes.

2. Epidemiology

Estimations from the World Health Organisation indicate that the prevalence of COPD is widely under-recognised, and consequently prevalence rates vary widely across different studies. This is in part due to the nature of the disease which can develop insidiously, meaning individuals may not present with symptoms until the disease is in its advanced stages.

In Ireland, the Irish Thoracic Society suggest that approximately 440,000 people have COPD, with the condition thought be the second most common respiratory disease, accounting for a quarter of all deaths in Ireland (approximately 7,000 per annum) (Irish Thoracic Society, 2008).

Many estimates of the prevalence of COPD only include older adults (aged>30) in the studies. However, the INHALE report (Ireland Needs Healthier Airways and Lungs also notes that the prevalence of COPD in Ireland in younger adults (20-44 years of age) is relatively high in Ireland at approximately 5% (De Marco et al, 2004).

In the UK, prevalence rates also vary widely. The 2006 Healthcare Commission Report 'Clearing the air: A national study of chronic obstructive pulmonary disease' suggests that whilst 900,000 individuals have been diagnosed with COPD in the UK, a further 2 million plus individuals have undiagnosed COPD resulting in a true prevalence figure of approximately 3 million.

Figures from the United States indicate that the prevalence of COPD is rising rapidly, with an increase of over 40% in the last two decades. Estimated prevalence in the US is 1-3% in Caucasian women, and 4-6% in Caucasian men (BMJ Best Practice, 2009).

COPD becomes more common with increasing age. The condition is more common in low socio-economic groups, and the prevalence has increased in women over the last decade – both reflections of the patterns of tobacco use (heavy smoking has increased in prevalence in women over this time for example) (NCGC, 2009).

Worldwide estimates are that the prevalence of COPD is rapidly rising, as is the mortality rate from the condition. This is largely due to increases in tobacco use, especially among women and adolescents (Mannino, 2002).

3. Aetiology

COPD is virtually always a condition which occurs in response to the inhalation of tobacco smoke, although exposure to occupational substances and pollution are also risk factors (see ***Appendix A***). Studies suggest that approximately 10-25% of smokers will develop COPD to some extent (Løkke et al, 2006). Passive smoking is also a contributing factor (Silverman and Speizer, 1996).

COPD occurs due to a combination of small airway disease (obstructive broncholitis) and airway and parenchymal damage and disease (emphysema) which results in an inflammatory response and airway obstruction (GOLD, 2009). Airway obstruction is defined as:

- FEV1 (forced expiratory volume in 1 second) post-bronchodilator as <80% of predicted value and
- FEV1/FVC ratio (forced expiratory volume in 1 second / forced vital capacity) of <0.7.

The contributing effects of the small airway disease and parenchymal damage vary from individual to individual, resulting in a disease path that whilst usually progressive, can vary considerably in each sufferer.

In a very small number of cases there is a genetic link affecting causality (American Thoracic Society, 1995). The inherited deficiency of anti-protease enzyme alpha1-antitrypsin is associated with development of COPD. The affected gene has been identified and a number of variants described. 95% of people with severe deficiency have a greatly increased risk of emphysema especially in smokers. [2]

4. Diagnosis

There is no specific clinical test which will diagnose COPD. The diagnosis is made using clinical judgement to assess the history of symptoms and physical examination factors along with spirometry tests to assess the degree of airway obstruction which is present.

A diagnosis of COPD should be considered in any patient who smokes, and who presents with dyspnoea, chronic cough or sputum production.

4.1 Overview

Studies suggest that many cases of COPD are not diagnosed. This is due to a number of factors, including the behaviour of the condition itself – symptoms may not become apparent until the condition is in more advanced stages, and individuals may not seek treatment at early stages either due to the fact that the condition is not well known - therefore an individual may not recognise the severity of the symptoms, or due to reluctance to seek help as they do not wish to cease smoking.

4.2 Clinical Features

COPD should be considered if the following clinical features are present (GOLD, 2009, NCGC, 2009):

- **Dyspnoea** that is progressive, persistent, exacerbated with exercise
- **Chronic cough** that can be intermittent and unproductive
- **Chronic sputum production**
- **History of exposure to risk Factors:**
  - Tobacco Smoke
  - Occupational Chemicals, Occupational dust (see ***Appendix A)***
  - Smoke (usually non-developed world from indoor cooking)
- Confirmation of **Airway obstruction** using spirometry, where the airway limitation is not fully reversible.
- A history of frequent ‘winter’ bronchitis
- Wheeze

Individuals with COPD may also present with an acute onset of breathlessness, fever and chest pain if an infectious component is involved in an acute exacerbation.

The degree of breathlessness can be assessed using the Medical Research Council

Dyspnoea Scale which describes a framework to categorise the degree of breathlessness experienced by an individual with COPD. Evidence suggests (Ozalevli and Ucan, 2006) that this scale is the most accurate and appropriate scale for measuring breathlessness for COPD.

Grade	Degree of breathlessness related to activities
1	Not troubled by breathlessness except on strenuous exercise
2	Short of breath when hurrying or walking up a slight hill
3	Walks slower than contemporaries on level ground because of breathlessness, or has to stop for breath when walking at own pace
4	Stops for breath after walking about 100m or after a few minutes on level ground
5	Too breathless to leave the house, or breathless when dressing or undressing

Table 5: Medical Research Council Dyspnoea Scale (adapted from Fletcher, 1959)

4.3 Other History

Other signs and symptoms that the individual may report include the following:

- weight loss
- exercise intolerance
- Disturbed sleep pattern, fatigue
- Ankle oedema

Any Symptoms of chest pain or haemoptysis should be assessed. These symptoms are not common in COPD and suggest an alternate diagnosis should be considered (NCGC, 2009).

4.4 Physical Examination

4.4.1 Observation

The degree of functional impairment that an individual is experiencing can be assessed to some extent through informal observation of the individual during the consultation process. Symptoms of exercise intolerance such as mouth or purse lip

breathing, hyper-inflated chest and/or the use of accessory muscles to aid respiration are not present at rest unless COPD is advanced; early stages of the disease may have little or no observable clinical signs present.

Other observable signs may include peripheral oedema, cyanosis etc.

A sub-group of individuals do not have good ventilator drive and tend to become drowsy and cyanosed with right ventricular failure (RVF) and peripheral oedema in the later stages of the disease, (formerly known as “blue bloaters”). The terms “blue bloater” and “pink puffer” are now rarely used and have little relevance to diagnosis or the assessment of disability

4.4.2 Physical Examination

There may be no abnormalities in the early stages of the disease, with abnormal clinical findings only becoming apparent as the disease progresses.

Respiratory Features

In COPD clinical features primarily affect the respiratory system. Physical features that may be found include a “barrel shaped” chest with increased AP diameter, held in the position of near full inflation. The shoulders are held in a “shrugged” attitude. Reduced chest expansion may be noted (i.e. the change between full inspiration and expiration is less than the expected 5cm (average in a male)). Resonance may be increased on percussion leading to ‘drum-like’ sounds due to hyper-inflation of the chest.

On auscultation, breath sounds may be quieter than normal due to reduced airflow and the expiration phase may be prolonged. There may be added wheezy sounds (high pitched expiratory rhonchi)

Abdominal palpation

The overexpansion of the lungs may make the liver appear larger by downward displacement.

Cardiovascular Features

Central cyanosis from polycythaemia and hypoxia may be present. Progressive lung damage results in pulmonary hypertension. This may progress to signs of right ventricular failure with raised jugular venous pressure (J.V.P.), peripheral oedema, right parasternal pulsation from a hypertrophied right ventricle, increased splitting of the 2nd heart sound and true hepatomegaly (cor pulmonale).

Systemic Features

With severe COPD many patients show evidence of poor nutrition, muscle wasting and weight loss, though this may be masked by the development of peripheral oedema.

4.5 Investigations

4.5.1 Spirometry

The fundamental diagnostic confirmation for COPD is spirometry which is used to demonstrate the presence of airway obstruction. Airway Obstruction is considered to be present when:

- *Post-Bronchodilator* $FEV_1/FVC < 0.7$ (i.e. 70%) and $FEV_1 < 80\%$ predicted value (GOLD, 2009).

Peak flow measurement is no longer recommended as this may significantly underestimate any airway obstruction which is present (Irish Thoracic Society, 2005; NCGC, 2009), meaning that a patient with COPD may have a normal peak flow reading (Nolan, White and Pearson, 1999).

Spirometry testing alone will not provide sufficient information to differentiate COPD from asthma. The differentiation was traditionally provided by reversibility testing – the use of spirometry in conjunction with a short-acting bronchodilator to indicate the degree of reversibility of airway obstruction, however recent evidence indicates that reversibility testing does not provide information to support the differentiation of asthma and COPD. This is due to a number of factors. Repeated tests can show fluctuations which may vary from small fluctuations on one day to large changes in magnitude between tests performed on different days. The degree of change which is required to be considered as reversibility has not been formally documented and is therefore arbitrary, and an individual may use different techniques or inspiratory movements on repeated or subsequent tests that will affect the result. The differentiation of COPD from asthma, and confirmation of a COPD diagnosis should be made on clinical history and presenting features (NCGC, 2009). (NOTE: NEW RECOMMENDATION IN THE 2010 NCGC GUIDELINES)

Whilst spirometry does not accurately predict disability or the degree of functional impairment that an individual may experience as a result of their COPD, it does provide information to indicate probable prognosis of the condition (Anthonisen et al, 1996; Jones, 2001). Spirometry also detects airway limitation before an individual experiences any symptoms from COPD. This early detection accompanied by lifestyle interventions such as cessation of smoking may mean that more severe aspects of the disease do not develop (Irish Thoracic Society, 2005).

It should be noted however that spirometry results may not provide accurate information to support a diagnosis of COPD in the elderly (where lung function may fall below the predicted due to other causes), or in the young (where a clinical picture of symptomatic COPD is present but lung function has not been sufficiently impaired to result in FEV1 being lower than 80% of the predicted value (NCGC, 2009). In these two groups, alternative diagnosis should be considered, but further investigations may be required to confirm a diagnosis of COPD (NCGC, 2009) (NOTE: NEW RECOMMENDATION IN THE 2010 NICE GUIDELINES)

Guidelines on the performance and interpretation of spirometry are available from the Irish Thoracic Society at the following link: http://www.irishthoracicsociety.com/documents/SpirometryGuidelinespdf.pdf (Link

accessed January 2010).

4.5.2 Classification of Breathlessness from Spirometry Results

There are a number of different classifications quantifying the degree of airway obstruction determined by spirometry results. Whilst it is recommended that the degree of severity of impairment to an individual is assessed using lung function measures in conjunction with other indicators such as quality of life, functional impairment and exercise tolerance; spirometry result classification may also provide information pertinent to ongoing treatment and prediction of prognosis.

The Irish Thoracic Society (2005) recommends that COPD severity from Spirometry results are classified using the GOLD (2009) classification system for airway obstruction:

- Stage 1: **Mild COPD** - Mild airflow limitation - $FEV_1/FVC < 0.7$ (i.e. 70%) and $FEV_1 \geq 80\%$ predicted value. Individuals with this stage of COPD may not be aware they have the disease. Cough or sputum production may be present.
- Stage 2: **Moderate COPD** - Worsening airflow limitation – FEV_1/FVC under 70% and FEV_1 50% to 80% of predicted. This is the stage that individuals will most commonly seek medical advice
- Stage 3: **Severe COPD** – Further worsening of airflow limitation - FEV_1/FVC under 70% and FEV_1 30% to 50% of predicted. Individuals will experience greater functional impairment, increased breathlessness and repeated exacerbations of their COPD.
- Stage 4: **Very Severe COPD** – Sever airflow limitation FEV_1/FVC under 70% and FEV_1 under 30% of predicted or FEV_1 under 50%, with chronic respiratory failure (if present, stage 4 COPD may be diagnosed even if FEV_1 is 30% of predicted. Functional impairment at this stage is considerable and the condition (exacerbations) is potentially fatal.

Note: Stage 0 which appeared in earlier guidelines is not included in recent versions of the GOLD classification.

4.5.3 Other Investigations

Other investigations which are required as a component of the diagnosis of COPD include:

- Chest X-Ray to exclude other differential diagnoses
- Full Blood Count
- Calculation of body mass index.

Additional investigations which may be performed include (NCGC, 2009):

- Alpha-1 antitrypsin if exposure to risk factors is not present or family history

is indicative of this genetic abnormality (referral to a specialist centre may be required)

- Serial Peak Flow measurements – to exclude asthma should uncertainty remain – serial measurements should show some degree of variation, and should show a response to treatment initiation with bronchodilators. In COPD serial measurements will show a progressive decline rather than variability.
- Pulse Oximetry – if indicated by physical symptoms such as cyanosis
- Sputum culture – if an infections component is suspected either as a differential diagnosis or as the underlying cause of a COPD exacerbation.

4.6 Table of diagnostic features and terms

Term	Definition	Diagnostic criteria
Chronic Bronchitis	Cough and sputum for 3 months in 2 successive years	History of symptoms
Airways obstruction	Diffuse peripheral airway narrowing with increased resistance to airflow	↓ FEV_1 ↓PEF
Asthma	Reversible airways obstruction with airway inflammation and hyper-responsiveness	Bronchodilator and steroid response
Emphysema	Dilatation of the terminal airspaces with destruction of alveoli	Histopathology CT scan ↓K_{CO}
Respiratory failure	Failure to maintain arterial oxygen and CO_2 tensions	↓ P_aO_2 ↑PCO_2
Cor pulmonale	Chronic lung disease causing pulmonary hypertension and leading to right heart hypertrophy.	Oedema ↑JVP ECG Echocardiography
Effort tolerance	Maximum energy expenditure	Measured in METs VO_2 max

5. Differential Diagnosis and Comorbidity

All of the symptoms of COPD are not specific to this condition alone, and can occur in a number of other disorders. Whilst it is important to ensure that other causes of breathlessness and productive cough should be excluded, it should be noted that a number of these conditions can commonly exist alongside COPD, particularly in aging individuals or those who have a long history of smoking.

5.1 Clinical Features Which Differentiate COPD and Asthma

The most important differential diagnosis is confirmation of COPD and exclusion of Asthma.

The differentiating features are in the history and in the investigations.

The clinical features which differentiate COPD from Asthma are shown in the table below.

FEATURE	COPD	Asthma
Smoker or ex-smoker	Nearly all	Possibly
Symptoms under age 35	Rare	Common
Chronic productive cough	Common	Uncommon
Breathlessness Persistent and progressive	Common	Variable
Night time waking with breathlessness and/or wheeze	Uncommon	Common
Significant diurnal or day to day variability of symptoms	Uncommon	Common
Day to day variation in PEF	Minimal	Usual
Response to bronchodilators	Poor	Good

Table xx: Features of COPD and Asthma (adapted from NCGC, 2009)

5.2 Other Differential Diagnoses of COPD

Other differential diagnoses to COPD are listed below.

5.2.1 Breathlessness Due to Respiratory Centre Stimulation by Hypoxia or Excess CO_2

Common Causes

- Pulmonary oedema
- Pulmonary embolus

- Pneumothorax
- Pneumonia
- Lobar collapse
- Pulmonary fibrosis
- Anaemia

Uncommon causes

- Acidosis
- Pregnancy
- Cyanotic congenital heart disease
- High altitude
- Arteriovenous fistula

5.2.2 Breathlessness Associated with an Increased Work of Breathing (Obstructive Ventilatory Defects)

Common Causes

- Asthma
- Bronchiectasis
- Cystic fibrosis

Uncommon Causes

- Upper airways obstruction
- Byssinosis

5.2.3 Other Common Causes

- Sarcoidosis
- Fibrosing alveolitis
- Extrinsic allergic alveolitis
- Pneumoconioses
- Large pleural effusion
- Extensive lung resection

- Chest wall deformity. Scoliosis etc.
- Pulmonary oedema
- Left ventricular dysfunction

5.2.4 Other Uncommon Causes

- Large tumours
- Large hiatus hernia
- Lymphangitis carcinomatosa
- Connective tissue diseases
- Aspiration pneumonitis
- Infections

5.2.5 Conditions associated with Decreased Neuromuscular Power

These conditions are all relatively uncommon

More Common

- Myasthenia gravis
- Polyneuritis

Less Common

- Poliomyelitis
- Motor neurone disease
- Muscular dystrophies

5.3 Comorbidity

COPD is a condition which is often co-exists alongside other smoking related diseases such as ischaemic heart disease (NCCCC, 2004; NCGC, 2009).

Other comorbidities which commonly affect COPD include allergies, asthma and poor nutritional status.

6. Treatment

6.1 Treatment Options for Chronic Obstructive Pulmonary Disease

It is important to note that effective treatment can considerably benefit an individual with COPD in helping improve and control their symptoms. To this extent, recent guidelines suggest that COPD should be thought of as a treatable disease. However, it should also be noted that COPD is a progressive disease, and even with the best treatment lung function will worsen over time (GOLD, 2009). Individuals with COPD should be made aware that treatment is ongoing, and will not be discontinued once symptom control is achieved.

6.1.1 Treatment Goals

There are a number of treatment goals which should be achieved for effective treatment of COPD. These include:

- Relieve symptoms
- Prevention of disease progression
- Improvement in exercise tolerance
- Improvement in general health
- Appropriate referral of individuals who will benefit from pulmonary rehabilitation
- Prevention of complications, rapid recognition and treatment of any complications which do occur
- Prevention of exacerbations of the condition and rapid recognition and treatment of any exacerbations which do occur
- Reduction in risk of mortality
- Recognition of end stage COPD and the initiation of effective palliative care.

(Gold, 2009; NHS Institute for Innovation and Improvement, 2009)

6.2 Self Management Plan

It is recognised that with many chronic conditions, individuals who have an effective self management plan are able to manage their condition more effectively, and respond to exacerbations of their condition more rapidly. A self management plan is a cornerstone of treatment for COPD as with the treatment of asthma. However, unlike asthma, (where the evidence suggests that the use of management plans by

individuals improves control of the condition and reduces exacerbations and the need for unplanned treatment) the evidence regarding the use of management plans for COPD suggests that although they support the rapid recognition of an exacerbation, the use of a management plan by an individual does not appear to improve outcomes, reduce the need for emergency treatment or reduce the number of exacerbations an individual may experience (McGeoch et al, 2006; Wood-Baker et al, 2006, NHS Institute for Innovation and Improvement, 2009).

Management plans for COPD should provide individualised information which covers the following areas:

- **Assess and Monitor Disease:**
- **Management of Stable COPD** – details of the individual's usual medication routine, follow up appointments etc.
- **Manage an Exacerbation:** Recognition of early signs of an exacerbation or worsening COPD symptoms (breathlessness, more sputum, coloured sputum, and/or fever) and actions should be taken should an exacerbation occur:
  - How to initially increase the use of short-acting bronchodilators, and if there is no response, when to contact a primary healthcare professional.
  - Indications as to when antibiotic treatment may be required (e.g. if sputum becomes discoloured and/or increases in volume)
  - Who to contact and what to do in an emergency situation
- **Reduction of Risk Factors**:
  - Lifestyle and medication issues which should be implemented to prevent exacerbations.
  - Smoking cessation techniques and support
  - General lifestyle advice e.g. general health, diet and exercise.

(Gold, 2009; NHS Institute for Innovation and Improvement, 2009

6.3 Non Pharmacological Therapies

There are a number of non-pharmacological therapies which may be of benefit to an individual with COPD. These include:

- Psychosocial plus pharmacological interventions for smoking cessation
- Psychosocial plus pharmacological interventions for smoking cessation
- Pulmonary rehabilitation

- General physical activity
- Inspiratory muscle training
- Peripheral muscle training

6.3.1 Smoking Cessation

Smoking cessation in individuals with COPD is the single most important intervention that can be taken to help control their disease. The importance of stopping (or reducing, if an individual absolutely cannot stop) smoking cannot be underestimated. Smoking is associated with a continuation in decline of lung function, whereas cessation of smoking slows the rate of decline in lung function to that of a non-smoking individual in a relatively short space of time, therefore having important benefits in terms of ongoing morbidity and mortality.

This protocol does not specifically cover therapies which can be used to help support an individual who wishes to stop smoking. Details of technology appraisals of various smoking cessation methods can be found at the UK National Institute for Clinical Excellence Website www.nice.org.uk.

6.3.2 Pulmonary Rehabilitation

Pulmonary Rehabilitation comprises of a multidisciplinary programme of care for patients with chronic respiratory impairment. The programme provides rehabilitation covering physical exercise, disease education, diet and nutrition advice, and psychological and behavioural techniques which are tailored to the individual's needs and circumstances to help increase respiratory function. Many patients with COPD start to avoid exercise due to their breathlessness. The condition can also lead to depression, de-motivation and social isolation. It is more suitable for individuals with moderate to severe forms of the disease but not for individuals who are unable to exercise.

There is some evidence that pulmonary rehabilitation leads to reduced admissions and ongoing care, and it is considered to be a cost effective method of treatment, however it has limitations in that despite good availability in North America and Western Europe, pulmonary rehabilitation service availability is more limited in the UK and Ireland.

(NHS Institute for Innovation and Improvement, 2009; NCGC, 2009).

6.3.3 Surgery

Surgical interventions (bullectomy, lung volume reduction surgery or lung transplant) can be beneficial to selected patients who have not shown a response to other measures. It should be noted however, that such procedures are expensive, and are often only indicated when an individual's COPD has reached the point of severity that palliative measures are considered (GOLD, 2009).

6.4 Pharmacological Therapies

The choice of pharmacological therapy is dependent on a number of different factors. Other indicators should be taken into account in addition to the degree of airway limitation which is detected using spirometry. These include the improvement in symptoms experienced by an individual in response to therapy, and how rapidity of the response; the degree of functional limitation experienced by the individual, their exercise tolerance levels, and their preferences regarding the mediation regimes and delivery systems. Cost and availability of specific therapies may also be an issue which may affect drug choice.

It should be noted that this protocol is not written with the intent of providing the level of detail necessary for determining treatment options for a specific individual but merely to provide an overview of the main treatment options available.

6.4.1 Step-Wise Treatment

As with asthma, COPD is usually treated with step-wise treatment. One treatment is commenced and evaluated, and if required, treatment can be 'stepped-up' to the next level either by using additional preparations, or changing to a different type of medication in order to achieve symptom control. Equally treatment can be 'stepped-down' when required, for example after an exacerbation.

This stepwise approach to therapy is recommended by the GOLD clinical guidelines (GOLD, 2009). The National Institute of Clinical Excellence Guidelines (NCCCC, 2004, NCGC, [draft], 2009) also include medication which changes according to the stability of COPD and the individual's symptoms and exacerbations however the regimes documented within each protocol differ slightly in terminology and in the selection of medication for each disease category. At the time of preparation of this protocol the 2004 NICE guidelines are in the process of update and are expected to be available in final form during 2010. The 2004 version of the NICE guideline for COPD, along with draft consultation documents for the 2010 update can be accessed at www.nice.org.

A working summary from the GOLD guidelines is provided for information purposes only below (Adapted from GOLD, 2009; NCGC 2009):

Stage 1 COPD: and initial treatment for individuals presenting with breathlessness and exercise intolerance - consider short-acting bronchodilators as a first-line treatment.

Stage 2 COPD: Consider the addition of long-acting bronchodilators (GOLD, 2009) in addition to short-acting bronchodilators. Consider pulmonary rehabilitation therapies.

Stage 3 COPD: Consider the addition of inhaled corticosteroid therapy to regular and as-required bronchodilators, if frequent exacerbations occur.

Stage 4 COPD: long-term oxygen therapy should be added to inhaled therapies, and possible surgical interventions should be considered.

Patient education, appropriate vaccinations and lifestyle interventions, particularly smoking cessation should be considered for every individual with COPD.

6.4.2 Bronchodilators

This group of drugs are fundamental in the treatment of COPD, either as a reliever to reduce symptoms on an as needed basis, or as a preventer in the form of a regular medication to prevent or control symptoms. Inhaled forms of bronchodilators are most commonly used, as this delivery route results in fewer side effects than medications administered orally, however patient education in effective techniques in the various delivery systems (e.g. inhalers or spacers) is vital to ensure maximum effectiveness.

Short Acting ß-Agonists

This group refers to the selective $ß_2$ agonists. Salbutamol (Ventolin) and Terbutaline (Bricanyl) are common examples. They are taken by inhalation, and have a rapid onset of action. (The effects begin after 15 minutes, and last about four hours.) The $ß_2$ agonists cause bronchodilation, and are used as used in COPD as an initial treatment, and also as a rescue therapy for individuals with more severe forms of COPD who require longer acting forms of bronchodilators as maintenance therapy.

Possible side effects include tremor, palpitations and muscle cramps, although these are uncommon when the drugs are taken in inhaled form.

Long Acting ß-Agonists

Examples include Salmeterol (Serevent) and Eformoterol (Oxis). They achieve their maximum effect in 2 hours and last for about 12 hours.

Anticholinergic Bronchodilators

The commonest example is Ipratropium Bromide (Atrovent). These drugs block the cholinergic bronchoconstrictor effect of the Vagus nerve. Maximum effect is achieved 30 – 60 minutes after use, and it lasts for about 4 hours. Anticholinergic bronchodilators are not as effective as ß-agonists, but are effective used in conjunction with ß-Agonists as a combination therapy if COPD symptoms are not adequately controlled, or exacerbations are frequent, without increasing the side effects of each class of drugs (BMJ Best Practice, 2009).

Research from the UPLIFT (Understanding Potential Long-term Impacts on Function with Tiotropium) Trial – one of the largest COPD trials undertaken, suggests that the Anticholinergic Bronchodilator Tiotropium is associated with sustained improvements in lung function, reduced hospitalisation for exacerbations, and increased quality of life and physical activity. This includes individuals with early stages of the disease, and those who have not ceased smoking. Lung function was not affected however. (Tashkin et al, 2008).

6.4.3 Inhaled Corticosteroids

Beclomethasone and budesonide are the most common examples of this class of

drug. Fluticasone is a more potent drug, and is used at half the dose. They are highly effective at reducing bronchial reactivity and inflammation and are used in individuals with COPD who have frequent exacerbations of their condition. The dosage required is usually much higher than the doses of inhaled corticosteroids required for control of asthma symptoms (NHS Institute for Innovation and Improvement, 2009). Peak effect usually occurs 3–7 days after initiation of treatment. Side effects are dose-dependent, inhaler device-dependent, and technique-dependent. In adults, adverse effects such as oropharyngeal candidiasis, sore throat, cough and thinning of the skin become more likely once a daily dose of 1000 mcgs of beclomethasone is reached.

Reversibility testing with oral corticosteroids is no longer necessary prior to starting a person on inhaled corticosteroids (NHS Institute for Innovation and Improvement, 2009).

Inhaled corticosteroids are only licensed in the UK for use in combination with a long-acting beta2-agonist. Recommendations for their use in clinical guidelines may not concur with this licensing (e.g. The National Institute for Health and Clinical Excellence (NICE) 2004 guidelines state that their recommendations for inhaled corticosteroid use is 'off licence' (NCCCC, 2004). In practice they are normally used with long-acting bronchodilators (NHS Institute for Innovation and Improvement, 2009).

6.4.4 Oral Corticosteroids

Oral corticosteriod treatment is not recommended in the treatment of individuals with COPD, but may be required to manage exacerbations of the condition. This group of drugs have a variety of adverse side effects, and so maintenance therapy is not usually recommended. The 2004 NICE guideline (NCCCC, 2004) does state however that there may be a small number of individuals for whom adequate symptom control cannot be achieved without using this for of therapy.

6.4.5 Theophyllines

These drugs are effective bronchodilators. They are taken orally as sustained release formulations. The theophyllines have a narrow therapeutic index, with considerable individual variation in the necessary dose. Therefore, it is necessary to monitor blood concentrations. For these reasons, they are used much less now that long acting ß-agonists are available. Common side effects include nausea and vomiting, abdominal discomfort, headache, malaise, tachycardia and fits. There are numerous drug interactions with other common treatments such as erythromycin, phenytoin and cimetidine.

6.4.6 Mucolytics

Mucolytics may be of use in individuals with COPD, particularly in individuals who have frequent exacerbations. A Cochrane review suggested there was a reduction of 20% in exacerbations when mucolytics were used (Poole and Black, 2006). Mucolytics are usually given over a 3-6 month period, however recent guidelines suggest the use of routine mucolytics is not recommended (GOLD, 2009).

6.4.7 Antibiotic Therapy

Antibiotic therapy is not routinely recommended for stable COPD as evidence indicates this provides no reduction in the number of exacerbations an individual has (Francis and Spicer, 1960; Francis, May and Spicer, 1961) but may be appropriate in the management of exacerbations (NHS Institute for Innovation and Improvement, 2009). The National Institute for Clinical Excellence 2004 guidelines recommend antibiotic therapy only should an individual present with increased purulent sputum production (NCCCC, 2004; NCGC, 2009) however other guidelines support a less stringent approach to antibiotic administration, suggesting that antibiotics may be appropriate should symptoms become more severe than normal (GOLD, 2009).

6.4.8 Oxygen therapy

Individuals with severe cases of COPD may develop hypoxaemia, which can result in symptoms of cor pulmonale, principally peripheral oedema. Once this occurs the prognosis is poor, untreated less than half of individuals will survive >5 years (NCGC, 2009). Oxygen can either be administered in long periods (for example overnight) or used as a reliever to lessen the effect of symptoms on exertion.

Long term oxygen therapy (therapy of >15, ideally 20 hours per day) should be considered for individuals severely affected by COPD who have an oxygen saturation of <92% breathing air.

Oxygen therapy is the only treatment that has been shown to improve survival times for COPD (GOLD, 2009)

6.4.9 Vaccinations

It is recommended that individuals with COPD have a yearly seasonal influenza and one-off pneumococcal vaccination. Evidence indicates that the seasonal influenza vaccination decreases COPD morbidity and mortality by 50% (Nichol et al, 1994, Wongsurakiat et al, 2003). Vaccinations are particularly effective in the elderly population.

6.5 End-Stage COPD - Palliative care

Palliative care should be considered in individuals with severe COPD which is continuing to deteriorate despite appropriate treatment, or is expected to die from their disease within the next year. This may be indicated by an increase in the frequency of admissions or exacerbations in an individual with severe disease (>2 per year) (NHS Institute for Innovation and Improvement, 2009).

7. Prognosis (Main Prognostic Factors)

The prognosis is profoundly influenced by smoking habit. Continuation of smoking in COPD leads to a continuous steady decline in lung function, early disability and reduced life expectancy. However, ceasing smoking or discontinuing exposure to other precipitating substances (for example an irritant in occupational exposure) has a significant positive effect on symptoms, and prevention or delay of airway limitation and a reduction in progression of the symptoms (GOLD, 2009). It is suggested that smoking cessation will reduce the rate of decline in lung function to that of non-smokers (Anthonisen et al, 1994). Subsequent risk of mortality from COPD is also reduced if smoking is ceased.

Pulmonary rehabilitation can lead to significantly improved effort tolerance in COPD patients, even though lung function tests are not improved (Donner and Muir, 1997, Ong et al, 2001). Rehabilitation in individuals with moderate symptoms is more successful than in individuals with severe symptoms (Wedzicha et al, 1998).

Prognosis figures in terms of survival rates are not available specifically for Ireland; however, the five-year survival rates from time of diagnosis of COPD for the UK are estimated as:

- In men with mild disease is 78%, which falls to 30% with severe disease.
- In women with mild disease is 72%, which falls to 24% with severe disease.

(Soriano et al, 2000):

Estimation of mortality rates, as with prevalence, is complex; with the true death rate difficult to ascertain (NCGC, 2009). This is due to a number of factors. Deaths may result from complications of COPD rather than the disease itself, or be certified as occurring due to a specific complication rather than the underlying disease. In addition, the individual may have died of a cause or condition not directly related to COPD but the condition may have had an effect on the comorbid disease. COPD is a common comorbidity in other smoking related diseases such as ischaemic heart disease and lung cancer for example (NCGC, 2009). Differences in diagnostic terms and varying diagnostic criteria from study to study also increase the variation in published prevalence and mortality rates.

The recently published INHALE report (Ireland Needs Healthier Airways and Lungs) states that for all respiratory disease, Ireland has the 2nd highest death rate in Europe (Kyrgyzstan being 1st), with deaths now exceeding the rate of deaths from coronary heart disease. This accounts for 1 in 5 of all deaths in Ireland, twice the European Average. 22% of these deaths are due to COPD. (Brennan, McCormack and O'Connor, 2008).

UK Mortality statistics suggest there are 30,000 deaths annually from COPD, 90% of these in individuals aged >65 (Healthcare Commission, 2006; NCGC, 2009). This mortality rate is also almost double the European average (NCGC, 2009) mirroring Ireland.

8. Information Gathering at the In Person Assessment

The primary disablement from COPD is due to reduced exercise tolerance.

Initially there is minimal disablement, which may only be apparent when running.

As the disease progresses there is limitation in walking quickly and climbing flights of stairs.

This progresses to limitation in walking at a normal pace and in climbing a flight of stairs.

Later the effort of mild exertion limits activities, such as dressing and undressing, washing, rising from sitting and walking even a few steps.

Eventually even minimal effort is not tolerated and there will be breathlessness at rest.

The gold standard for diagnosis of COPD is by spirometry. The diagnosis requires a post-bronchodilator FEV_1 of less than 80% of the predicted value accompanied by an FEV_1 / FVC ratio of less than 70%.

However, functional activity limitation (disability) does not directly correlate with FEV_1 measured/FEV_1 predicted (impairment) due to other factors such as body mass index, general level of fitness, and psychological factors.

Cardiopulmonary exercise testing is a better guide of disability although this is rarely performed except in experimental work (Orgeta et al, 1994).

Clinical examination findings do not correlate well with functional ability and the assessment of claimants is best made from the evidence of:

1. The History of Activities of Daily Living (Average Day) taking variation into account.

2. Informal Observation of the client's activities at examination.

3. Medication taken and attendance at Chest Clinic.

9. Analysis of Effect on Functional Ability

Eligibility to the Department of Social and Family Affairs various Illness-related schemes and the Activation Programme is determined primarily by the degree of Ability/Disability and its expected duration.

The degree of Ability/Disability assessed, using the following Indicators, can be depicted on the Ability/Disability Profile illustrated below.

9.1 Indicators of Ability/Disability

Normal

- No history of respiratory symptoms lasting more than three months in each of the preceding two years
- Not breathless on exertion
- On no relevant medication
- Never been referred to a chest clinic
- No breathlessness observed during examination
- Clinical examination normal

Mild

- Breathless on strenuous exercise
- Occasional use of short acting bronchodilators
- No chest clinic referral
- No breathlessness observed during the examination process
- Clinical findings normal

Moderate

- Breathless on walking 100m. Or climbing one flight of stairs at a normal pace
- Breathless on walking 100m at a slow pace or climbing one flight of stairs without stopping
- On occasions breathlessness prevents walking 100m at a slow pace or climbing one flight of stairs without stopping
- May attend chest clinic

- If available FEV1/FVC under 70% FEV1 50-80%
- On short and long acting bronchodilators
- May show some evidence of breathlessness during examination process
- Clinical findings may be within normal limits with perhaps some reduction of breath sounds

Severe

- Stops for breath after a few minutes on level ground
- May require supervision of assistance outdoors
- Breathlessness limits indoor activities
- Likely to attend chest clinic
- If available FEV1/FVC under 70% FEV1 30-50%
- Likely to be on short and long acting bronchodilators and inhaled corticosteroids
- May have required treatment with courses of oral steroids and antibiotics in the last year during acute exacerbations
- May show mouth or purse lip breathing during examination
- Cyanosis may be present
- Peripheral oedema may be evident
- Chest may be barrel shaped
- Chest expansion less than 5 cms
- Increased resonance
- Breath sounds reduced with a prolonged expiratory phase
- Possible high pitched expiratory rhonchi

Profound

- Too breathless to leave home
- May be virtually bed or wheelchair bound and dependant on carers for bodily care
- On maximum medication and domiciliary oxygen
- History of chest clinic attendance and likely to have required admission

during acute exacerbations

- FEV1/FVC under 70% FEV1 under 30% if available
- Clinical findings as in severe and in addition
- Central cyanosis
- Signs of Right Ventricular failure
- Also may show signs of poor nutrition, muscle wasting and weight loss

9.2 Ability/Disability Profile

Indicate the degree to which the Claimant's condition has affected their ability in ALL of the following areas.					
	Normal	Mild	Moderate	Severe	Profound
Mental health/Behaviour	☐	☐	☐	☐	☐
Learning/Intelligence	☐	☐	☐	☐	☐
Consciousness/Seizures	☐	☐	☐	☐	☐
Balance/Co-ordination	☐	☐	☐	☐	☐
Vision	☐	☐	☐	☐	☐
Hearing	☐	☐	☐	☐	☐
Speech	☐	☐	☐	☐	☐
Continence	☐	☐	☐	☐	☐
Reaching	☐	☐	☐	☐	☐
Manual dexterity	☐	☐	☐	☐	☐
Lifting/Carrying	☐	☐	☐	☐	☐
Bending/Kneeling/Squatting	☐	☐	☐	☐	☐
Sitting	☐	☐	☐	☐	☐
Standing	☐	☐	☐	☐	☐
Climbing stairs/Ladders	☐	☐	☐	☐	☐
Walking	☐	☐	☐	☐	☐

10. Summary of Scheme Criteria

Scheme eligibility criteria are maintained on the DSP website and are accessible from the following links:

- **Carer's Allowance**
  http://www.welfare.ie/EN/OperationalGuidelines/Pages/carers_all.aspx
- **Carer's Benefit**
  http://www.welfare.ie/EN/OperationalGuidelines/Pages/carers_ben.aspx
- **Disability Allowance**
  http://www.welfare.ie/EN/OperationalGuidelines/Pages/disall.aspx
- **Disablement Benefit**
  http://www.welfare.ie/EN/OperationalGuidelines/Pages/oib_disableb.aspx
- **Domiciliary Care Allowance**
  http://www.welfare.ie/EN/Schemes/IllnessDisabilityAndCaring/Carers/DomiciliaryCareAllowance/Pages/DomiciliaryCareAllowance.aspx
- **Illness Benefit**
  http://www.welfare.ie/EN/OperationalGuidelines/Pages/illben.aspx
- **Injury Benefit**
  http://www.welfare.ie/EN/OperationalGuidelines/Pages/oib_injuryb.aspx
- **Invalidity Pension**
  http://www.welfare.ie/EN/OperationalGuidelines/Pages/invalidity.aspx
- **Respite Care Grant**
  http://www.welfare.ie/EN/OperationalGuidelines/Pages/respitegrant.aspx

Appendix A - Occupational COPD

Studies have shown that up to 15% of cases of COPD could be linked to occupational exposure to irritants such as gasses or dusts (Blank and Toren, 2007).

A list of commonly affected occupations, and identified causative agents is provided below.

A.1 Occupations Linked to the Development of COPD

Occupations which have been linked to the development of COPD in a number of studies include the following industries:

- Coal or other forms of Mining (e.g. gold), Quarrying. This includes both underground and surface mining
- Asbestos cement production
- Iron and Steel
- Textiles, cotton manufacture
- Construction
- Agriculture
- Factories where exposure to dust, fibres or gasses is present
- Printing
- Electronics
- Leather production
- Plastic and rubber production
- Armed Forces
- Transport, trucking, automotive industries
- Health care
- Beauty e.g. exposure to dust from manicures; hairdressing
- Food products manufacturing
- Professional office cleaning services

(Marek and Zejda, 1998, Hnizdo et al, 2002; Industrial Injury Advisory Council, 2007; Rodriguez et al, 2008; Health and Safety Executive, 2009)

A.2 Occupational COPD – Potential Causative Agents

The following list details agents which have been suggested as a cause of occupational COPD:

- Dust for example:
  - coal dust
  - asbestos cement
  - grain
  - cotton
  - flour
  - Wood
- Fibres (e.g. textile fibres)
- Non-halogenated hydrocarbons, polycyclic aromatic hydrocarbons
- Paints
- Resins
- Varnishes
- Gasses (e.g. sulphur dioxide, oxides of nitrogen)
- Welding fumes
- Man-made vitreous fibres
- Minerals such as silica
- Isocyanates
- Cadmium
- Vanadium

(Marek and Zejda, 1998, Industrial Injury Advisory Council, 2007; Health and Safety Executive, 2009)

Appendix B - Professional and Patient Resources

The following list of resources is not intended to be an exhaustive list of all resources available but documents a variety of resources identified during the preparation of this protocol which may be of use.

B.1 Clinical Guidelines

The Global Initiative for Chronic Obstructive Lung Disease international guidelines which were revised in 2009 can be downloaded as a full report version, an executive summary or as a pocket version from the following link: http://www.goldcopd.com/GuidelinesResources.asp?l1=2&l2=0 (Link accessed January 2010)

Lothian Chronic Obstructive Disease (COPD) Guidelines: http://www.lothianrespiratorymcn.scot.nhs.uk/wp-content/uploads/2009/05/Lothian-COPD-Guidelines2.pdf (Link accessed January 2010)

National Institute of Clinical Excellence: 'Chronic Obstructive Pulmonary Disease: Full Guideline DRAFT' http://www.nice.org.uk/guidance/index.jsp?action=download&o=46089

B.2 Diagnostic Checklists

Diagnostic checklists from a range of international resources can be accessed at the International COPD Organisations Website – Professional Resource section. These include:

- Australian Lung Foundation Diagnostic Checklist which can be accessed at:http://www.internationalcopd.org/documents/English/COPD_Checklist.pdf (Link accessed January 2010).

B.3 Sample Management Plans

Resources for Sample Management Plans include:

- Australian Lung Foundation Example Management Plan which can be accessed at :http://www.internationalcopd.org/documents/English/COPD_Action_Plan.pdf (Link accessed December 2009)
- COPD Management Plan from the Asthma Foundation of New Zealand which can be accessed at http://www.asthmanz.co.nz/files/PDF-files/Resources/COPD_Management_Plan_14_October.pdf (Link accessed January 2010)
- Lothian Chronic Obstructive Disease (COPD) Guidelines: http://www.lothianrespiratorymcn.scot.nhs.uk/wp-

content/uploads/2009/05/Lothian-COPD-Guidelines2.pdf (Link accessed January 2010) (Sample Management Plan on P21)

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