

Practice *Nursing*

PROMOTING EXCELLENCE IN EDUCATION AND CLINICAL PRACTICE

Improving outcomes in COPD

*by Rachel Booker
with an editorial by Beverley Bostock-Cox*

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COPD is a slowly progressive, devastating disease. Owing to underdiagnosis many people cope with increasingly restrictive lung function which severely limits their quality of life over time. The pathological changes that occur in the lungs and airways in COPD are examined and their consequences explained.

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Nurses in general practice need to know how to recognise and assess the risk factors and symptoms of COPD, as an early diagnosis can substantially reduce morbidity and improve quality of life. The correct conduct and interpretation of spirometry are explained as part of the diagnostic process.

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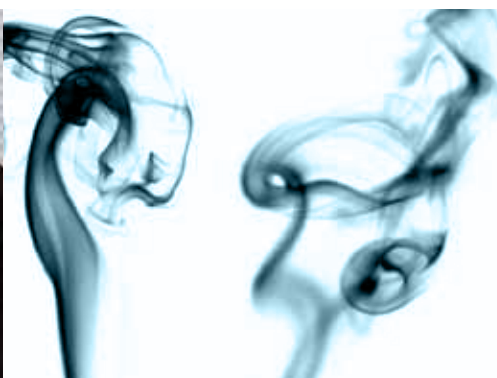
Realistic aims for COPD management include slowing or halting disease progression, relieving symptoms to reduce disability, and decreasing the frequency and severity of exacerbations. The use of a range of available medications is discussed and the importance of regular review emphasised.

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The need to know COPD

My Uncle Steven (his name has been changed to protect his modesty) called recently to inform me about his latest doctor's appointment. At 70-odd, he's a miracle of modern medicine: he has type 2 diabetes, coronary heart disease, arthritis and 'asthma'. He has a retail therapy habit to challenge my own and he still part-runs his own successful business. He told me that he's now got a new problem: 'It's called C-O-P-D,' he said. 'Do you know anything about it? I've never heard of it but at least it's not emphysema—I was worried it might be that.' There are tribes undiscovered who knew that Uncle Steven had COPD, not asthma, mainly owing to the insidious onset in middle age of cough, sputum and breathlessness on a background of significant smoking history. He is recalled regularly to the 'asthma' clinic, where he's kitted out with nebulas and oral steroids to keep him ticking over until next time. Is Uncle Steven unusual?

It is going to be an exciting year for nurses working in the respiratory field—most of us in general practice. Along with the updated Quality and Outcomes Framework (QOF) and revamped asthma guidelines from the British Thoracic Society, the first respiratory national service framework (NSF) is in the offing. The choice of COPD as the first respiratory NSF is no accident, as COPD costs society dearly, both in financial terms and in its impact on patients, their carers and families.

According to estimates from the Office of Health Economics, COPD will be the fourth leading cause of death worldwide and the fifth in more economically developed countries by the year 2030. The British Lung Foundation (BLF) (2007) estimates that 3.7 million people have COPD in the UK but only about 900,000 have been diagnosed and are receiving appropriate management.

Little wonder that we are being asked to focus on the prevention of COPD as well as its treatment and management. Yet how many of us feel up to this challenge? A survey by Education for Health showed that over half of the nurses running COPD clinics in general practice had no qualifications in COPD. The medico-legal implications of this are worrying. On the other hand, few in the general public understand what COPD is, why it happens and what can be done about it (BLF, 2006). Practice nurses are on the frontline of COPD management, and this affords important opportunities to anticipate and reduce the damage as early as possible.

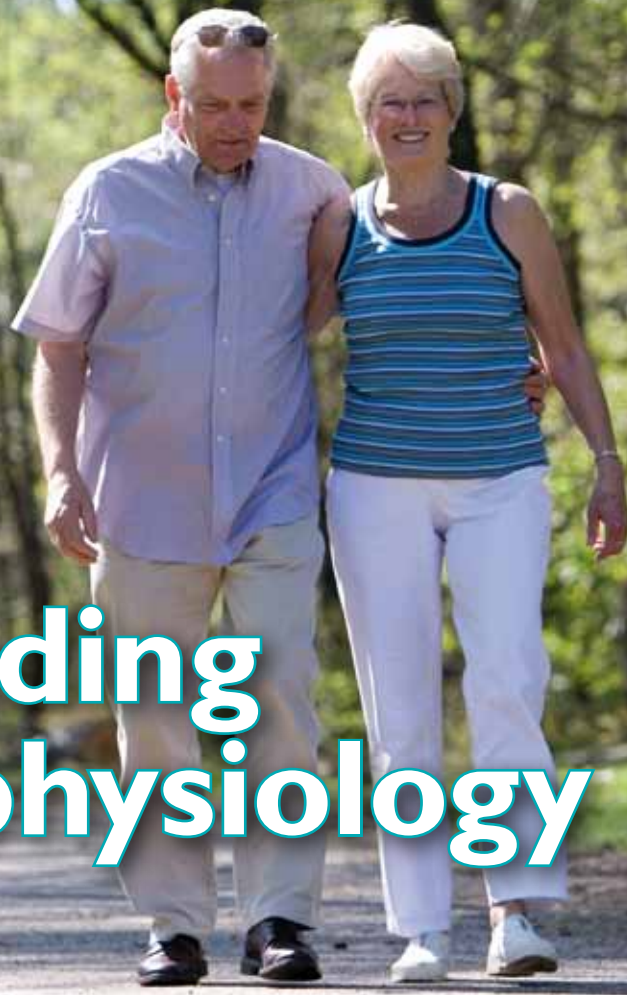
We need to understand the risk factors, right back to the antenatal period. We need to arm ourselves with the most effective smoking cessation tools. We need to know who to screen and when. We need to know how to screen—spirometry is only useful if it is done correctly. Once a diagnosis has been made, we need to know how and when to initiate treatment to optimise quality of life.

COPD is a multisystem disorder, affecting not only cardiorespiratory health but also the musculoskeletal system and cognitive ability. All of this, from a condition which, for the most part, is preventable.

In this special supplement of *Practice Nursing* we will learn how early prevention and recognition of COPD can improve both the significant morbidity and mortality associated with this disease. We will learn about the approaches, both pharmacological and non-pharmacological, which can have a significant impact on the lives of people with, or at risk of, COPD. I hope that you will enjoy these articles and, most importantly, that you will learn from them.

Practice nurses are on the frontline of COPD management, and this affords important opportunities to anticipate and reduce the damage as early as possible.

Understanding the pathophysiology



Chronic obstructive pulmonary disease (COPD) is a slowly progressive, devastating disease that causes 30,000 deaths a year. It is substantially underdiagnosed, leaving many people to cope with increasingly compromised lung function which, over time, severely limits all aspects of their quality of life. The pathological changes that occur in the lungs and airways in COPD, and their consequences are reviewed in this article.

COPD is slowly progressive and patients tend to present late. Disease progression is punctuated by increasingly frequent and severe exacerbations.

Chronic obstructive pulmonary disease (COPD) is characterised by airflow obstruction. It is defined by the Global Initiative for Chronic Obstructive Lung Disease (GOLD) (2007) as:

a disease state 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.

It is mainly caused by cigarette smoking. Reliable statistics of prevalence are hard to obtain. Soriano et al (2000) reported a physician-diagnosed prevalence of 1.7% of men and 1.4% of women, but it is widely acknowledged that COPD is underdiagnosed. Nacul et al (2007) estimated a prevalence of 5.3% in over-45-year-olds in the UK. Shahab

et al (2006) estimated spirometry-defined COPD in England at 13.3%.

COPD is important. Many patients have to retire prematurely and, as the disease progresses, become high users of scarce NHS resources. COPD accounted for 13% of all emergency hospital admissions in 2004 (British Thoracic Society (BTS), 2006), and it causes about 30,000 deaths annually.

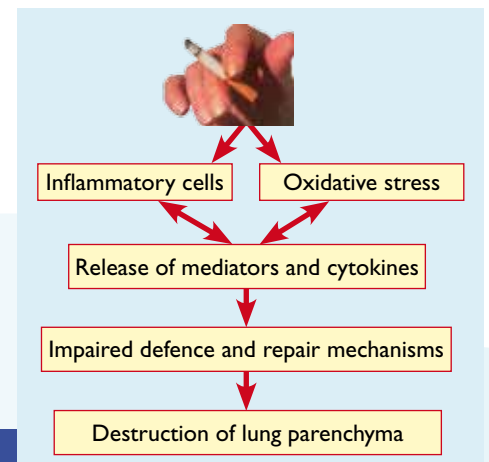


Figure 1. Pathogenesis of emphysema

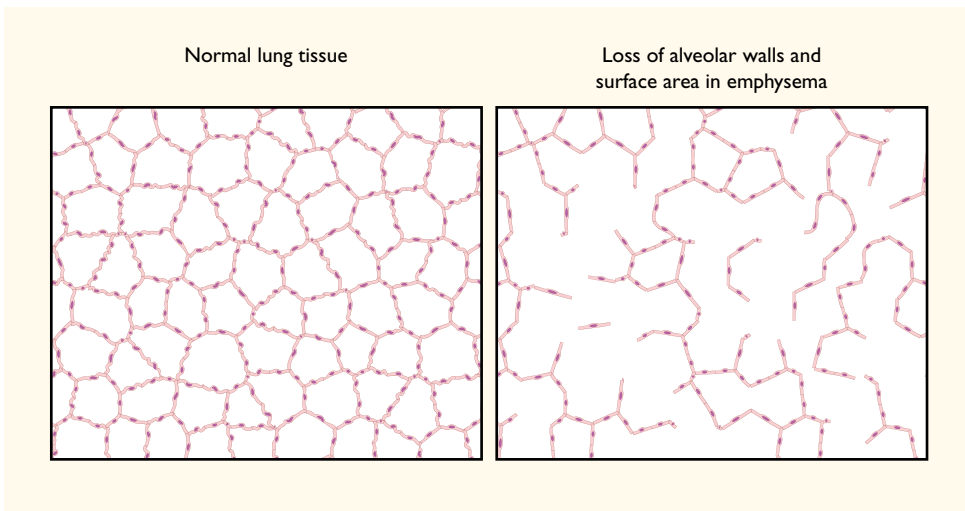


Figure 2: Cross-section of normal and emphysematous lung

is relatively well understood, but rare. Less is known about the development of emphysema in the majority of patients who are not α_1 AT deficient, but there appears to be a familial pattern and many patients report a positive family history (Wood and Stockley, 2006).

Loss of alveolar tissue has two main effects:

- Reduction of lung surface area (Figure 2) and disruption of gas exchange
- Airflow obstruction.

COPD is devastating. Slowly progressive breathlessness and increasingly frequent exacerbations eventually impact on every aspect of a patient's life. It causes financial hardship, progressive physical and emotional disability, disrupts relationships and places burdens on family and carers (British Lung Foundation (BLF), 2006).

Disease spectrum

COPD is an 'umbrella' term encompassing:

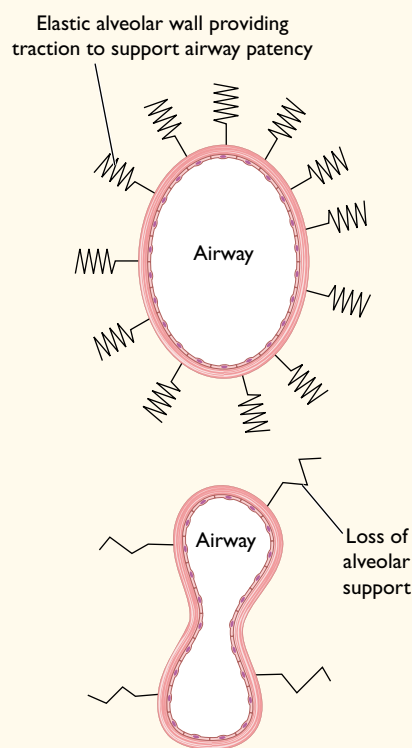
- Emphysema
- Chronic bronchitis (and small airway disease, also known as chronic bronchiolitis)
- Chronic severe asthma.

The clinical picture, and to some extent the outcome, varies depending on the balance of these three components. Many patients have elements of all three conditions.

Emphysema

Emphysema causes destruction of alveolar walls and, in severe forms, destruction of the terminal airways from which alveoli arise (Snider et al, 1985). It is a disease of smokers. Cigarette smoke attracts inflammatory cells, particularly macrophages and neutrophils, into the small airways and alveoli. These cells produce toxic mediators that destroy lung tissue and perpetuate the inflammatory process. Other constituents of tobacco smoke damage lung tissue directly, or disrupt the normal balance between damaging enzymes (proteases) and protective enzymes (anti-proteases). Cigarette smoke also contains oxidants that increase oxidative stress and inflammation (Spurzem and Rennard, 2005) (Figure 1).

Figure 3: Loss of alveolar tissue and airway obstruction



Proteases are normally inactivated or destroyed by anti-proteases. These enzymes form part of the lung's biological defence mechanisms. The most widely studied anti-protease is α_1 -antitrypsin (α_1 AT). Some individuals have a genetically acquired deficiency. They tend to have a strong family history of COPD and develop severe emphysema at an early age. Deficiency of α_1 AT

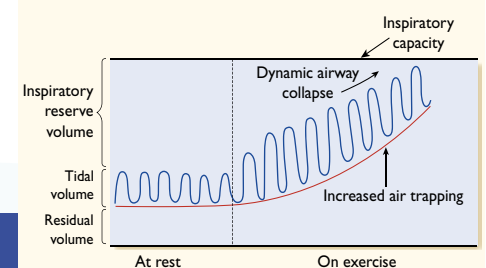
Figure 4. The effect of exercise on lung volumes in COPD

The natural elastic recoil of lung tissue provides traction and support to maintain the patency of small airways. This support is lost when alveolar tissue is destroyed. During exhalation, particularly forced exhalation during exercise, small airways are 'squeezed' causing them to narrow. In emphysema, when airways are unsupported, they narrow significantly and may collapse, trapping air in the lungs (Figure 3). Air trapping reduces the ability to respond to increased respiratory demand and further increases breathlessness (Figure 4).

Chronic bronchitis and bronchiolitis

Chronic bronchitis is defined as 'the production of phlegm on most days for three months during two consecutive years' (Medical Research Council, 1965). Cigarette smoke irritates the airways, altering the production and clearance of mucus. Mucus glands increase in size and goblet cells increase in number (Jeffery, 2000). Smoking also reduces the number and length of the cilia. Thus, the amount of mucus in the respiratory tract increases and the ability to clear it decreases. Pooling of mucus increases susceptibility to infection and produces an unpleasant and troublesome cough.

Excess mucus production is abnormal, but can occur without causing airflow obstruction. There are smokers with chronic



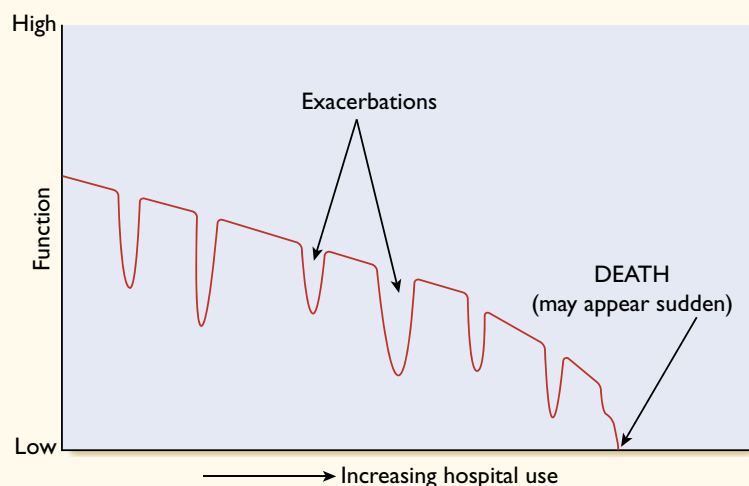


Figure 5. COPD disease trajectory

punctuated by increasingly frequent and severe exacerbations. Recovery is slow and frequent exacerbations accelerate the decline of lung function and reduce quality of life (Cote et al, 2007). COPD fits the ‘organ failure’ disease trajectory described by Gomes and Higginson (2006) (Figure 5).

It is difficult to predict prognosis in COPD with accuracy. Often patients are ‘brought back from the brink’ many times before eventually succumbing. Death, while appearing to be sudden, will be at the end of a progressive decline. Timing discussions about end-of-life care is problematic and often these discussions do not take place at all. Palliative care provision is limited and quality of life in the terminal phase is frequently poor (Gore et al, 2000). There is, however, much that can be done to improve both living and dying with COPD.

bronchitis who do not have airflow obstruction and do not fit the definition of COPD. The main site of mucus production is from mucus glands in the large airways. Excess mucus here causes productive cough but may not cause airflow obstruction.

Goblet cells are not normally found in airways less than 2 mm in diameter. A key feature of smoking-related small airway disease is mucus metaplasia: the presence of goblet cells in these airways. Mucus production at this site is thought to contribute to the airflow obstruction that is the key feature of COPD, but may not be associated with chronic productive cough.

Small airway disease affects airways of 2–3 mm diameter. This area of the lungs is sometimes called the ‘silent area’, since major structural changes can occur with little in the way of symptoms. As well as mucus metaplasia and mucus plugging, other structural changes include (Jeffery, 2000):

- Smooth muscle hypertrophy
- Oedema of the airway wall, and
- Fibrosis around the airway.

This leads to narrowing and distortion of the airway and airflow obstruction.

Asthma

A characteristic feature of asthma is variability and reversibility of airflow obstruction. However, as a result of long-standing inflammation and bronchospasm some patients will develop a degree of fixed airflow obstruction. This is likely to be most marked in those with more severe asthma (Vignola et al 2003). The risk is greatly increased by smoking.

Irreversible, structural changes in the airway include (Jeffery, 2000):

- Fibrosis and collagen deposition
- Thickening of the basement membrane
- Disruption of the epithelium
- Increased airway smooth muscle.

Non-smokers who develop COPD as a result of chronic severe asthma are likely to exhibit a degree of reversibility and variability in their symptoms, although reversibility will not be complete.

Symptoms and consequences

COPD causes:

- Breathlessness on exertion
- Chronic cough
- Chronic sputum production
- Wheeze
- Frequent winter ‘bronchitis’.

Progressive airflow obstruction and disruption of gas exchange eventually lead to chronic hypoxia and respiratory failure. The stage at which this occurs varies between individuals. Some patients appear to drive an increasing respiratory rate to compensate, becoming increasingly breathless but maintaining oxygen levels. They develop respiratory failure as a terminal event. Others appear to tolerate a degree of hypoxia without becoming unduly breathless. Chronic hypoxia, however, leads to complications involving the heart and other organs, and will shorten life.

Disease trajectory

COPD is slowly progressive and patients tend to present late. Disease progression is

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Making the diagnosis

Many people have no symptoms in the initial stages of COPD or their early symptoms may be overlooked or explained away as the effects of ageing. It is important that nurses in general practice know how to recognise and assess the risk factors and symptoms of COPD, as an early diagnosis can improve outcomes and quality of life. The conduct and accurate interpretation of spirometry is explained in this article.

Chronic obstructive pulmonary disease (COPD) is slowly progressive and patients tend to present late. Many attribute symptoms of early disease to increasing age, lack of general fitness or a 'normal' consequence of smoking. There is evidence to suggest that smokers avoid visiting their doctor with symptoms because they 'will only be told to stop smoking' (Halpin et al, 2002). Patients have commonly lost more than half their respiratory reserve before they present (Fletcher and Peto, 1977).

COPD is underdiagnosed, and many people remain untreated despite severe loss of lung function (Shahab et al, 2006). The first step to improving diagnosis rates is to recognise and assess individuals at risk of COPD. A thorough history needs to be taken and appropriate diagnostic tests carried out.

Risk factors

The most important risk factor for COPD is cigarette smoking. Other factors tend to be 'additive' to smoking. COPD can occur in

non-smokers, but this is rare. Risk factors can be usefully separated into two groups:

- ▶ Those that impair lung growth, preventing the attainment of full, potential lung function
- ▶ Factors that accelerate lung function decline in adult life.

Rapid lung growth occurs prenatally and in early childhood, with a further 'spurt' during adolescence. Maximum lung function is achieved in early adult life. After the age of 25 years lung function normally begins to decline.

Reduced lung growth and development

Low birth weight (dysmaturity) is a risk factor for reduced lung function in adult life (Barker et al, 1991). Impaired antenatal lung growth has a long-term effect on lung structure and function (Stick, 2000). Prematurity alone does not seem to have a significant effect, although tiny infants, requiring long-term ventilation may be at risk. Further studies of these babies are needed as they age.

Maternal smoking is a risk factor for COPD and needs to be strongly discouraged. It is a major risk factor for:

- ▶ Low birth weight
- ▶ Reduced lung function at birth
- ▶ Increased risk of lower respiratory tract infection in early childhood
- ▶ The development of childhood asthma and atopy
- ▶ Worsening progress and outlook of childhood asthma.

All of these are risk factors for COPD in adult life (Young et al, 2000; Sethi, 2000; von Mutius, 2002).

Adolescence is a further period of rapid lung growth and is also the time when young people begin to smoke. Most regular smokers begin their habit in their early teens.

Accelerated lung function decline

From early adulthood, lung function loss normally occurs at the rate of 25–30 ml of forced expired volume in 1 second (FEV₁)

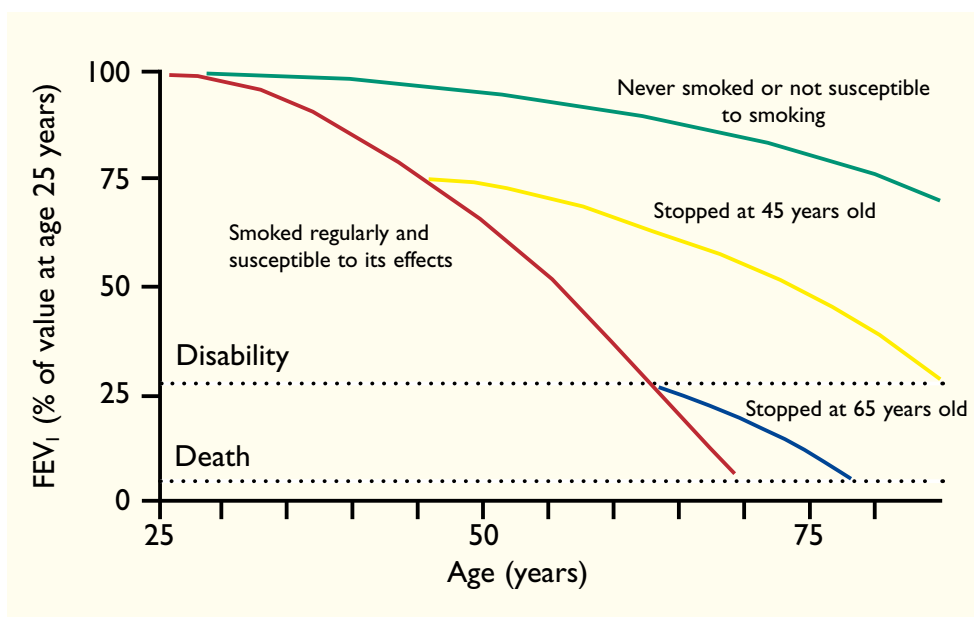


Figure 1. The effect of smoking on the rate of FEV₁ decline in susceptible individuals (Fletcher and Peto, 1977).

Assessment

Smoking history

Exposure to cigarettes needs to be quantified. The most widely recognised measure is the 'pack-year': 20 cigarettes a day equates to 1 pack year. Smoking one 'pack' a day for one year is 1 pack-year, two 'packs' a day for 1 year is 2 pack-years, thus:

$$\frac{\text{Cigarettes smoked/day}}{20} \times \text{Years smoked}$$

Pack-year calculation does not need to be exact, but it gives a good estimation of whether an individual is at risk or not. Exposure to more than 20 pack-years is significant for COPD (Barnes, 1999).

Occupational history

Many patients will have retired, but it is important to determine whether they have ever been exposed to occupational risk factors for both COPD and asthma.

Family history

Families often share life-style factors, e.g. smoking, but there is also likely to be a genetic element to susceptibility to COPD (Wood and Stockley, 2006). It is therefore important to ask if there is a family history of COPD, or other chest disease.

Asthma is a major differential diagnosis so it is important to ask if there is any family history of this, or other atopic illness such as hay fever or eczema. If so, asthma needs to be considered.

A familial element to cardiovascular disease (CVD) also needs to be considered.

Previous medical history

Cardiac disease and COPD cause similar symptoms. A previous history of rheumatic heart disease should raise the possibility of valve disease. CVD is a common comorbidity in COPD patients. A previous history of coronary heart disease, angina, dyslipidaemia or hypertension should raise suspicion and will require further investigation. Patients with diabetes are at high risk of CVD and need particularly careful evaluation.

per year. In susceptible smokers, this loss is accelerated to 50–60 ml FEV₁ annually (Figure 1). Estimates of how many smokers are susceptible to COPD vary from 15% (Peña et al, 2000) to 25% (Løkke et al, 2006) and 20–30% (Young et al, 2007).

Occupational exposure to high levels of organic and inorganic dusts and fumes, is associated with an increased risk of COPD (GOLD, 2007). In the UK coal mining is a recognised occupational risk for which compensation may be payable.

Occupational asthma is often not recognised and continued exposure to triggers can result in severe, irreversible asthma, indistinguishable from COPD. Smoking increases the risk of sensitization to occupational triggers and occupational asthma (Siracusa et al, 2006).

COPD is more common in socio-economically disadvantaged groups (Prescott et al, 2003). These groups are more likely to work in high-risk occupations and smoking rates are higher. However, the reasons for the high rates of COPD in these groups are multi-factorial and difficult to decipher. Poor nutrition and housing may also be important.

Indoor and outdoor air pollution have been extensively studied as risk factors for COPD, and evidence is now emerging. Poor air quality may reduce lung growth and accelerate decline (Viegi et al, 2006).

Symptoms

COPD causes progressive, non-variable symptoms. Patients do not have symptom-free days and experience daily symptoms that worsen during exacerbations (Bellamy

and Booker, 2004: 28–31). Symptom variability, with some symptom-free days, is characteristic of asthma.

Exertional breathlessness is a key symptom of COPD. However, its onset is insidious and patients adapt their lives to accommodate it, sometimes unconsciously. It is worth asking specific questions, e.g:

- ▶ Are you able to walk up two or three flights of stairs without stopping to get your breath?
- ▶ If you walk with a friend the same age, are you able to keep up, or do they have to slow down?
- ▶ When you are walking, can you carry on a conversation at the same time?

Enquiry should be made about productive cough. In COPD cough is usually worse first thing in the morning, but clears quickly with the production of grey or mucoid sputum. Sputum becomes discoloured and increases in volume during exacerbations. The chronic production of purulent sputum and/or frequent exacerbations should prompt investigation for bronchiectasis. A cough that wakes the patient at night is a key feature of asthma. Wheeze is non-specific and occurs in both asthma and COPD.

Regular 'winter bronchitis' is common in COPD. Patients have often attended every winter for a number of years before diagnosis. This represents a missed opportunity and the possibility of COPD should be considered in all smokers who attend with chest infections. They need to be followed up and offered spirometry when they are well.

Table 1. Key features differentiating asthma and COPD

	Asthma	COPD
Current or ex-smoker: >20 pack years	Possibly	Nearly all
Symptoms under the age of 35 years	Common	Uncommon
Chronic productive cough	Uncommon and variable	Common and persistent
Breathlessness	Variable day to day and diurnally	Persistent and progressive
Nocturnal waking with breathlessness or wheeze	Common	Uncommon
Family or personal history of atopy	Common	Uncommon

A childhood history of asthma, recurrent 'chestiness' or wheezing may indicate the recurrence of childhood asthma. There are a number of key features in the history that can help differentiate asthma from COPD (Table 1).

Differential diagnosis

COPD is a disease of older smokers. It is vital to ensure that alternative and comorbid conditions are recognised. A number of tests are recommended (NCC-CC, 2004).

A chest X-ray should be performed in all patients to help exclude an alternative diagnosis. It will not confirm COPD, but may show the hyperinflation typical of airway obstruction. Cardiac disease is less likely if the heart is a normal size and most, but not all, lung cancers will be visible on the X-ray by the time they cause symptoms. Chest X-ray is not reliable for diagnosing bronchiectasis. This requires referral for a high-resolution CT scan.

A full blood count should be taken to exclude anaemia. If the history suggests cardiac disease, an electrocardiogram, or referral for echocardiogram should be considered.

Spirometry

Spirometry is essential to confirm the presence and determine the severity of the airflow obstruction (NCC-CC, 2004)

Table 2. Severity of airflow obstruction

Post-bronchodilator FEV ₁	Severity of obstruction
50–79% predicted	Mild
30–49% predicted	Moderate
<30% predicted	Severe

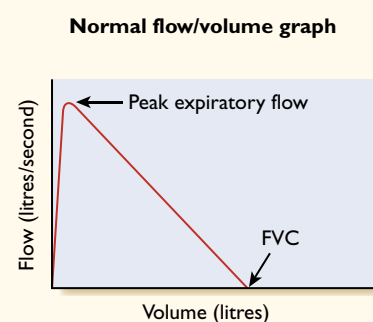
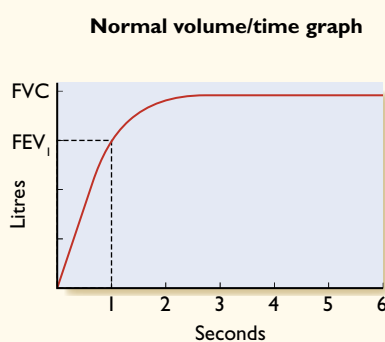
(Table 2). Miller et al (2005a) defined the essential lung volume measurements:

- **Relaxed vital capacity (VC or RVC):** 'The maximum volume of air that can be expired from the lungs during a relaxed, but complete expiration from a position of full inspiration'
- **Forced vital capacity (FVC):** 'The maximum volume of air that can be expired from the lungs during a forced and complete expiration from a position of full inspiration'
- **Forced expired volume in 1 second (FEV₁):** 'The maximum volume of air that can be expelled from the lungs in the first second of a forced expiration from a position of full inspiration.'

Miller et al (2005a) defined the airflow measurements as follows:

- **Ratio of FEV₁ to FVC (FEV₁/FVC, or FEV₁%):** 'The amount of air blown out in the first second of a forced expiration from a position of maximal inspiration expressed as a percentage of the total amount expired (regardless of time) during that forced manoeuvre'.

Figure 2. Normal volume/time and flow/volume graphs



If the VC is greater than the FVC, Miller et al (2005a) recommended calculating:

- **Ratio of FEV₁ to VC:** 'The amount of air expired during the first second of a forced expiration from a position of maximal inspiration expressed as a percentage of the total amount expired during a relaxed vital capacity manoeuvre'.

Airway obstruction can cause air trapping during forced expiratory manoeuvres. If the VC is greater than the FVC, the FEV₁/VC is a better indicator of the degree of obstruction than the FEV₁/FVC.

VC, FVC and FEV₁ are expressed as volumes (in litres) and as a percentage of the reference (predicted) value for someone of the same age, height, gender and ethnicity. The FEV₁/FVC and FEV₁/VC are expressed as a ratio or percentage.

The forced expiratory manoeuvre is also presented graphically as the volume/time and flow/volume trace. These graphs are essential for checking technical acceptability (Miller et al, 2005a). The volume/time trace is a graph of the volume exhaled against the time taken to exhale fully. Volume is on the vertical axis and time on the horizontal. The flow/volume trace is an additional or alternative method of displaying data from a forced exhalation. It is a graph of expiratory flow rate against the volume exhaled. The flow rate is on the vertical axis and the volume on the horizontal. Normal volume/time and flow/volume graphs have a typical shape and are shown in Figure 2.

In a healthy individual, lung volumes (VC, FVC and FEV₁) will be greater than 80% of the reference value. When airways are not obstructed about three quarters of the FVC can be exhaled in the first second of a forced exhalation, i.e. the FEV₁/FVC will be about 0.75, or 75%.

Table 3. Parameters of lung function in different defects

	Normal spirometry	Obstruction	Severe obstruction	Restriction
FVC	>80% of reference value	>80% of reference value	<80% of reference value	<80% of reference value
FEV ₁	>80% reference value	<80% of reference value	<80% of reference value	<80% of reference value
FEV ₁ /FVC	>0.7 (>70%) and usually about 0.75 (75%)	<0.7 (70%)	<0.7 (70%)	>0.75 (>75%) and often >0.8–0.85 (>80–85%)

Airflow obstruction reduces the volume of air exhaled in the first second of a forced exhalation, i.e. it will reduce the volume of the FEV₁ to less than 80% of the reference value and reduce the ratio of FEV₁ to FVC and VC. A ratio of less than 0.7 (70%) is indicative of airflow obstruction. The FVC is normal in mild to moderate airflow obstruction, but air trapping in severe obstruction can reduce it. *Table 3* summarises the effect of a variety of ventilatory defects on the parameters of lung function. *Figure 3* shows the typical shapes of the volume/time and flow/volume graphs in mild/moderate and severe airflow obstruction.

Conducting spirometry

Spirometry is hard work for patients and instruction and support from an adequately trained person is necessary. Spirometry is useless and potentially misleading if it is not conducted and interpreted properly. It is essential that the person conducting the test is appropriately trained, able to identify and correct technical errors, and maintains his/her skills with regular practice (Miller et al, 2005b). Anybody responsible for interpreting results must be trained, and ideally certified as competent for the task.

The spirometer calibration must be checked with a calibration syringe, and recorded on a daily basis. The accuracy must

also be verified regularly, the spirometer disinfected and cleaned appropriately, and the equipment maintained and serviced according to the manufacturer's recommendations (Miller et al, 2005b).

Bronchodilators need to be withdrawn before diagnostic spirometry (*Table 4*). It is also helpful to give some instructions to help patients prepare (*Figure 4*). Height (without shoes) and weight must be accurately measured. For safety reasons patients must sit for forced expiratory manoeuvres. Nose clips are used for relaxed expiratory manoeuvres but are not essential for forced manoeuvres, unless there is difficulty obtaining reproducible measurements.

The test must be explained carefully, the manoeuvres demonstrated and the patient encouraged. The role of 'coach' is vital. Maximum inhalation is needed at the start of each manoeuvre and every last drop of air has to be exhaled. For forced manoeuvres, vigorous encouragement to make a maximum effort, from beginning to end of the manoeuvre, is essential.

To ensure reproducibility a minimum of three relaxed and three forced manoeuvres are needed. They must be technically acceptable and the best two efforts should be within 5% or 100 ml of each other. If necessary up to eight forced manoeuvres can be undertaken in a single session, but no more (Miller et al, 2005a).

Reversibility testing

The main aim of reversibility testing is to help differentiate COPD and asthma. Potential for misinterpretation of the results and consequent misdiagnosis is great, however, because:

- There is no international agreement about what constitutes a 'positive' reversibility test
- The values and percentages suggested in guidelines are arbitrary and not based on research
- The extent of reversibility varies within an individual from day to day, such that 'positive' reversibility may be exhibited one day and 'negative' reversibility the next (Calverley et al, 2003)

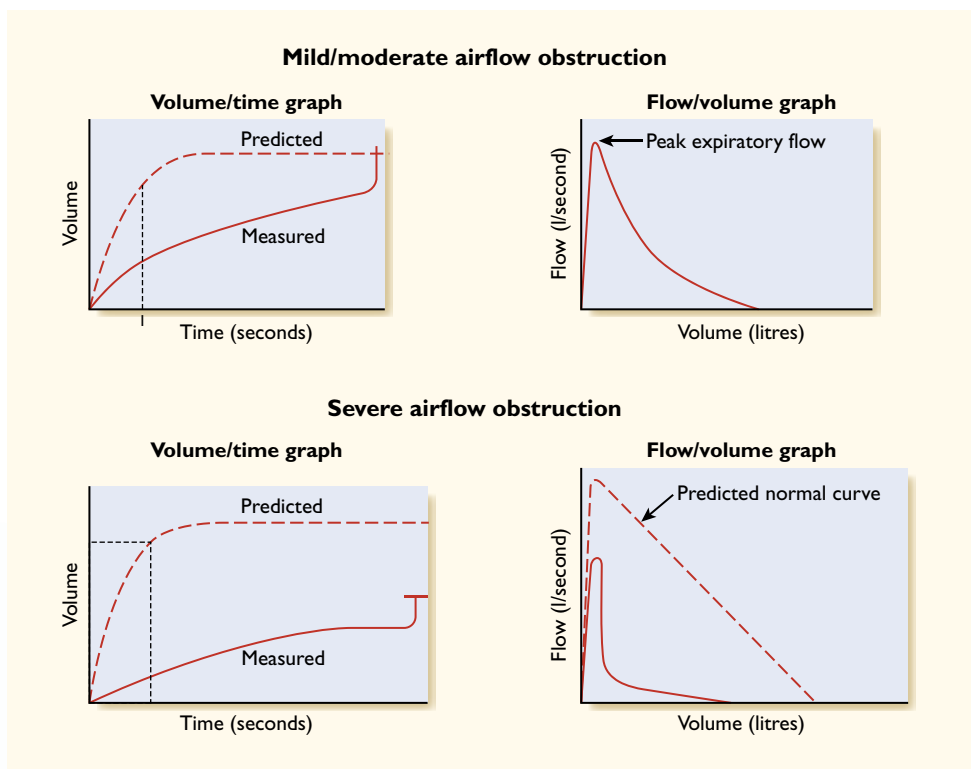


Figure 3. Typical shapes of volume/time and flow/volume graphs in mild/moderate and severe airflow obstruction

Table 4. Withholding bronchodilators before diagnostic spirometry and reversibility testing

Drug class	Example	Withhold before spirometry
Short-acting β_2 -agonists	Salbutamol, terbutaline	4 hours
Long-acting inhaled β_2 -agonists	Salmeterol, formoterol	12–24 hours
Long-acting oral β_2 -agonists	Oral salbutamol	12 hours
Short-acting anticholinergics	Ipratropium bromide	6 hours
Long-acting anticholinergics	Tiotropium bromide	24–36 hours
Sustained release theophyllines	Slo-phyllin, Neulin SA, Uniphyllin continuous	24–36 hours

- Reversibility testing gives no indication of which therapies will benefit the patient in the long term (NCC-CC, 2004).

A 'positive' reversibility test is not necessarily diagnostic of asthma. A large degree of reversibility, e.g. 400 ml of FEV₁, as suggested by NICE (NCC-CC, 2004), would make it more likely. However, some patients with asthma demonstrate less reversibility than this, and some with COPD show more.

The diagnosis of both COPD and asthma should only be made after careful consideration of:

- The clinical history
- The presentation
- The physical examination
- Spirometry
- Reversibility testing (if conducted).

Conclusions

If the burden of COPD is to be reduced, it is important to diagnose it as early as possible, and gently—but persistently—persuade and support patients to give up smoking. There are also effective pharmacological and non-pharmacological therapies that can

Figure 4. Patient information and preparation for spirometry

You have an appointment for some breathing tests on:

There are some things that we would like you to do, to help us get the most information out of the test.

If possible:

- Avoid eating a large meal within 2 hours of your appointment
- Do not smoke within 1 hour of your appointment
- Do not drink alcoholic drinks within 4 hours of your appointment
- Wear loose, comfortable clothing that does not restrict your breathing
- Do not take vigorous exercise within 30 minutes of the test
- Try to arrive for the appointment in plenty of time so that you have time to relax and visit the toilet.

Please let us know before the test if you have been unable to follow any of these instructions.

Thank you.

reduce symptoms and disability and improve quality of life and outcomes. The first steps to ensuring the best possible outcome, for patient and health service, are to:

- Think of COPD as a possibility when risk factors and/or symptoms are present
- Take a thorough history
- Perform the right diagnostic tests.

This will help confirm a diagnosis and enable the right treatment to be given at the earliest possible opportunity.

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Managing stable COPD

Realistic aims for COPD management include slowing or halting disease progression, symptom relief to reduce disability, and decreasing the frequency and severity of exacerbations. A range of medications available to treat COPD are discussed in this article and the steps necessary to gain the optimal benefits of medication are explained. The importance of regular review is emphasised.

It is only in the last 50 years that the aetiology and pathophysiology of COPD have been researched. The progressive, irreversible nature of COPD led to the mistaken belief that little could be done (Rennard, 2004). A 'blame culture' often exists because COPD is related to smoking, and patients may feel they do not deserve good treatment for a 'self-inflicted' illness. This view is sometimes reinforced by unsympathetic attitudes from relatives and, unfortunately, some health professionals. COPD affects the elderly and the poor, those least able to demand high standards of care.

Such attitudes are unjustifiable. Realistic aims for long-term management are to:

- Slow down or halt disease progression
- Achieve best possible symptom relief, thereby reducing disability
- Reduce the frequency and severity of exacerbations.

Nurses' role

Early diagnosis is required to achieve these aims. Regular review is also needed to ensure that appropriate interventions are offered, education and information needs are met, and symptoms are as well controlled as possible.

The management of long-term conditions such as COPD is well suited to nurse-led care, provided that the nurse is adequately trained. The diagnosis and management of COPD is often complex; patients are older and co-morbidity is common. High quality training and continuing professional development, together with support from medical colleagues and the multi-disciplinary team help ensure good quality care.

Disease progression

Smoking cessation is currently the only intervention proven to slow the progression of COPD. Accelerated FEV₁ decline in an 'at-risk' smoker can be reduced to that of a non-smoker or non-susceptible smoker at any stage in the disease process (Fletcher and Peto, 1977). The earlier a smoker stops the better, but it is never too late.

Health professionals should raise the subject of smoking cessation at every encounter. Doing so may be enough to:

- Prompt a committed smoker to think about stopping
- Encourage someone contemplating stopping to make a serious quit attempt.

No opportunity to support smoking cessation should be missed.

The pharmacological aids for smoking cessation endorsed by NICE (2008) are nicotine replacement therapy, bupropion and varenicline. Smokers who want to quit should be encouraged to use them as they improve the chances of success. Smokers who want to stop but, despite support, are unable to quit, should be referred to specialist services (British Thoracic Society (BTS), 2000).

Symptom reduction

Progressive breathlessness and chronic productive cough are distressing and disabling. The main approaches to reducing these symptoms are:

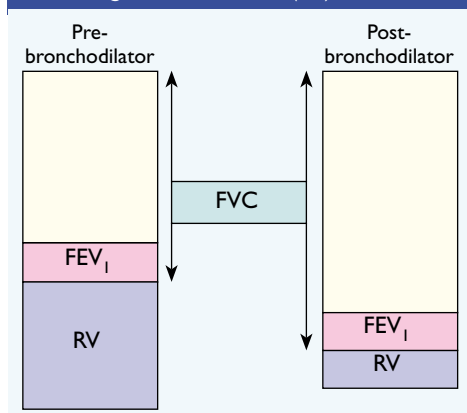
- Bronchodilators (long- and short-acting)
- Mucolytics
- Pulmonary rehabilitation.

Bronchodilators

Bronchodilators have only a modest effect on FEV₁ in COPD, but can decrease bronchomotor tone and airway resistance (Bellamy and Hutchison, 1981), reducing hyperinflation of the lungs and making



Figure 1. The action of bronchodilators in reducing residual volume (RV) in COPD



respiratory muscles more efficient (Figure 1) (O'Donnell and Webb, 1993). Breathing becomes easier and more comfortable, and exercise tolerance improves. They are therefore the mainstay of pharmacotherapy to improve breathlessness and exercise capacity.

The main classes of bronchodilators used in COPD are anticholinergics and beta₂ (β₂) agonists. Oral methylxanthines can be tried, but their potential to cause side effects and the need to regularly monitor serum levels has consigned them to third-line therapy (NCC-CC, 2004).

Anticholinergic bronchodilators have a particular role. Increased cholinergic tone is the major reversible component of airflow obstruction in COPD (Barnes, 2004). Short-acting β₂-agonists have a rapid onset of action, making them particularly suitable for immediate symptom relief.

Therapeutic response to different agents varies from person to person and should be assessed after 4–6 weeks of treatment in terms of symptomatic improvement. Five simple questions (Table 1) or the COPD

Table 1. Five questions to assess response to bronchodilators

1. Has your treatment made a difference to you?
2. Is your breathing easier in any way?
3. Can you do some things now that you couldn't do before, or can you do the same things, but faster?
4. Can you do the same things as before, but are now less breathless when you do them?
5. Has your sleep improved?

From: Jones et al, 2001

Table 2. MRC Dyspnoea Scale

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

Assessment Test (CAT)TM (www.catestonline.org) are practical for primary care use.

Evidence for the benefit of long-acting bronchodilators—both the long-acting anti-cholinergic, tiotropium, and the long-acting β₂-agonists, salmeterol and formoterol—is now strong. Long-acting bronchodilators produce sustained reduction in air trapping, which may explain their superior efficacy over short-acting agents in terms of symptom relief and improved lung function (Ramirez-Venegas et al, 1997; Celli et al, 2003). Inhalers combining long-acting β₂-agonists and long-acting anticholinergics are being developed and, like combinations of short-acting agents, are likely to be effective (Combivent Inhalation Aerosol Study Group, 1994).

Long-acting bronchodilators also produce improvements in health status and reduce the frequency and severity of exacerbations (Cazzola et al, 1995; Boyd et al, 1997; Ramirez-Venegas et al, 1997; Casaburi et al, 2002; Hanania et al, 2005; Niewoehner et al, 2005; Barr et al, 2006). Neither of these outcomes have been demonstrated with regular short-acting bronchodilators.

The UPLIFT trial, an important long-term study of tiotropium in COPD, demonstrated a reduced risk of exacerbations, their related hospitalisations and respiratory failure, as well as sustained improvements in quality of life (Tashkin et al, 2008). Benefits were also demonstrated for the combination salmeterol and fluticasone inhaler in the TORCH study (Calverley et al, 2007).

The current NICE guideline recommends short-acting bronchodilators as first line treatment for mild disease (NCC-CC 2004). However, mild disease can be associated with severe disability. Furthermore, most patients do not present until they have moderate or severe disease. Many newly diagnosed patients are symptomatic on a

daily basis, scoring 3 or more on the Medical Research Council (MRC) dyspnoea scale (Table 2). Therapy needs to start at a level appropriate to the severity of the symptoms and the disability they cause. An updated NICE guideline for COPD will be published in June 2010.

Mucolytics

Mucolytics reduce the viscosity of sputum, making it easier to clear. A recent review shows that they can significantly reduce the number of exacerbations and improve symptoms of chronic cough and sputum production in COPD (Poole, 2006).

The currently available agents for long-term use are carbocysteine and mecysteine. They are recommended for patients with chronic productive cough and should be continued if they produce symptomatic improvement (NCC-CC, 2004).

Pulmonary rehabilitation

Many patients cope with breathlessness by avoiding exercise. Sadly, loss of general fitness, with increasing breathlessness and disability is often the consequence. Patients lose confidence and functional ability, entering a downward spiral of disability, loss of self-esteem, social isolation and depression. The aim of pulmonary rehabilitation is to reverse this. It is defined by the NCC-CC (2004) as:

A multidisciplinary programme of care for patients with chronic respiratory symptoms that is individually tailored and designed to optimise each patient's physical and social performance and autonomy.

The cornerstone is individually prescribed exercise, i.e. aerobic training to recondition skeletal muscles and improve endurance, and exercise for specific muscle groups to improve

Table 3. Response to exacerbations

Symptoms	Action
Worsening breathlessness	<ul style="list-style-type: none"> Increased doses, or increased frequency of short-acting bronchodilators If breathlessness interferes with normal daily activity, short course of oral corticosteroids (prednisolone 30 mg daily for 7–14 days)
Increased sputum production and purulence	<ul style="list-style-type: none"> Short course of antibiotics, according to local guidelines for community-acquired infections Antibiotics are not indicated unless there is increased sputum purulence

From: National Collaborating Centre for Chronic Conditions, 2004

functional ability. This is undertaken in supervised group sessions twice weekly for 6–12 weeks. Patients are also given an exercise routine to continue at home, between visits, and after the programme finishes. Education to increase patients' and carers' understanding of COPD and its management is the other important component.

Pulmonary rehabilitation can have a number of important benefits (Stewart et al, 2001; Lacasse et al, 2007):

- Improved exercise performance
- Reduced breathlessness
- Improved health-related quality of life and health status
- Reduced hospitalisations and health service use.

Health economic research has demonstrated its cost effectiveness (Griffiths et al, 2001).

NICE (NCC-CC, 2004) recommends that pulmonary rehabilitation should be locally available and offered to all appropriate patients who consider themselves functionally disabled by COPD (a score on the MRC dyspnoea scale of 3 or more).

Pulmonary rehabilitation needs a patient's commitment and motivation. Patients also need to be on optimal medical treatment and able to exercise. Those with comorbid cardiovascular disease, or neurological or musculoskeletal problems that limit their ability to exercise may not be suitable.

Reducing exacerbations

Exacerbations are distressing and disabling, accelerate disease progression (Donaldson et al, 2002) and result in significant mortality. A UK audit demonstrated that, of 1400 acute COPD admissions, 34% were readmitted and 14% had died within 3 months (Roberts et al, 2002).

An exacerbation is defined as 'a sustained worsening of the patient's symptoms from their usual stable state which is beyond normal day-to-day variations, and is acute in onset' (NCC-CC, 2004).

Early recognition of deterioration with rapid, appropriate response is an essential

part of educating patients in self-management and may help reduce exacerbation severity, and give patients some control over the illness (Table 3). Patients need clear, written advice and need to know when to seek medical help. Some benefit from keeping emergency courses of antibiotics and corticosteroids at home to initiate prompt treatment.

It is important to ask patients how many exacerbations they experience. Patients who exacerbate frequently (i.e. two or more times a year) may benefit from long-acting bronchodilators or inhaled corticosteroids.

Inhaled corticosteroids

There have been large, long-term, placebo-controlled trials of inhaled corticosteroids in COPD (Pauwels et al, 1999; Vestbo et al, 1999; Burge et al, 2000; The Lung Health Study Research Group, 2000). None of these studies showed a significant reduction in lung function decline, and their use for disease modification cannot be justified. However, the ISOLDE trial (Burge et al, 2000) demonstrated that fluticasone propionate 1000 µg/day significantly reduced exacerbation rates ($P=0.026$) and associated decline in health-related quality of life in patients with moderate and severe COPD.

NICE (NCC-CC, 2004) recommends inhaled corticosteroids for patients who:

- Have an FEV₁ equal to or less than 50% reference value
- Have had two or more exacerbations requiring antibiotics or oral corticosteroids in a 12-month period.

The minimum daily dose of inhaled corticosteroid needed to reduce exacerbation frequency has yet to be determined. A meta-analysis suggests that moderate-to-high doses are needed (van Grunsven et al, 1999), but the NICE guideline makes no recommendation. In addition, none of the inhaled corticosteroids is licensed as a single agent for use in COPD; they are only licensed for use in combination with long-acting β_2 -agonists.

Combination inhalers

Long-acting bronchodilators and inhaled corticosteroids both reduce exacerbation frequency, and NICE recommends that when inhaled corticosteroids are prescribed they should be added to a long-acting bronchodilator (NCC-CC, 2004). The choice of bronchodilator—a long-acting β_2 -agonist, e.g. formoterol or salmeterol, or a long-acting anticholinergic, e.g. tiotropium—depends on the patient's response. Two combination inhalers are currently available:

- Formoterol 12 µg and budesonide 400 µg as the Symbicort 400 Turbohaler
- Salmeterol 50 µg and fluticasone 500 µg as the Seretide 500 Accuhaler.

It is interesting to note that Symbicort and Seretide are equally effective, despite different doses of corticosteroid. Dose ranging studies are needed.

Combination inhalers of long-acting anticholinergics and corticosteroids are being developed. In-vitro studies (Johnson, 2005) suggest there may be an interaction between corticosteroids and muscarinic receptors, making such a combination potentially useful. Further trials are needed. 'Triple' combination inhalers containing a corticosteroid, long-acting β_2 -agonist and a long-acting anticholinergic are also in development.

Inhalers

Inhaled therapy is preferable to oral. It is generally given at lower doses, is more effective and causes fewer side effects. Diminishing cognitive ability and manual dexterity may, however, make inhalers more difficult to master and the simpler the inhaler, the better. Patients' lifestyles and personal preferences need to be considered. Inhalers need to be discreet and portable for use outside the home to help patients remain socially active. Large volume spacers may not be the ideal solution.

Inhaler selection needs to take many factors into consideration and once taught, inhaler technique must be regularly checked.

Conclusions

Enormous progress has been made in the understanding of COPD management since the middle of the 20th century, and there is a

great deal of worldwide interest and research. Interest in COPD in the UK rose from a low level with the publication of the first national BTS guideline in 1997, and has been further raised with NICE guidance and the inclusion of COPD in the Quality and Outcomes Framework of the GMS contract. It is hoped that the development of a national clinical strategy, due for publication in 2010, will further improve service provision and reduce inequality of care.

While COPD still places a considerable burden on patients, their carers and society, the prospects are looking brighter. COPD has been described as the 'Cinderella' respiratory disease, but the likelihood of Cinderella going to the ball is improving.

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Griffiths TL, Phillips CJ, Davies S, Burr NL, Campbell

Martin started smoking when he was 14 years old and has made several unsuccessful attempts to stop over the past 30 years. At the age of 44 he has a successful but stressful career and finds it difficult to relax. He came to the surgery as he had developed a 'chest infection' which he attributed to working outside during the cold winter months.

He has complained of winter bronchitis for 2 years and has requested antibiotics, which were not prescribed as a viral infection was suspected.

He attends the nurse in a first-contact clinic. He describes symptoms of chronic cough and some shortness of breath with intermittent 'chest infections'. The nurse assesses his smoking history as over 20 pack-years, putting him at significant risk of COPD. It seems likely to the nurse that Martin is demonstrating some of the symptoms of early COPD.

Spirometry shows an FEV₁/FVC ratio of 64% with an FEV₁ of 71%, indicating obstructive lung disease, according to GOLD guidelines. With Martin's symptoms, this would suggest a diagnosis of mild COPD.

Martin is surprised to hear this. He was aware of the term COPD as his father has been diagnosed recently, but he is shocked

Case Study



that he might have it so young. He is also stunned to read on the spirometry printout that his lung age is estimated as 72 years old. Although these readings are not entirely reliable, Martin is concerned enough to ask what he can do about his new diagnosis.

This is a perfect opportunity for the nurse to explain to Martin the link between smoking and COPD. Using a combination of behavioural support and pharmacological interventions based on Martin's previous attempts at quitting and current NICE guidelines, the nurse can help Martin stop smoking, to slow the progression of his COPD.

Annual spirometry as part of a COPD review to monitor his FEV₁, along with any symptoms, will determine his need for COPD medication. The primary objective of COPD management is to minimise symptoms through the use of bronchodilators, particularly in the early stages.

Early detection of his COPD and quitting smoking successfully will mean that Martin's long-term outlook is much improved.

by Bev Cox

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Prescribing Information (UK)

SPIRIVA® (tiotropium)

Long acting, specific antimuscarinic agent, available as hard capsules of powder for inhalation, containing tiotropium bromide monohydrate equivalent to 18 micrograms tiotropium. Indication: Tiotropium is indicated as a maintenance bronchodilator treatment to relieve symptoms of patients with chronic obstructive pulmonary disease (COPD). Dose and Administration: Adults only age 18 years or over: Inhalation of the contents of one capsule once daily from the HandiHaler® device. Contraindications: Hypersensitivity to tiotropium bromide, atropine or its derivatives, or to the excipient lactose monohydrate which contains milk protein. Warnings and Precautions: Not for the initial treatment of acute episodes of bronchospasm, i.e. rescue therapy. Immediate hypersensitivity reactions may occur after administration of tiotropium bromide inhalation powder. Caution in patients with narrow-angle glaucoma, prostatic hyperplasia or bladder-neck obstruction. Inhaled medicines may cause inhalation-induced bronchospasm. In patients with moderate to severe renal impairment tiotropium bromide should be used only if the expected benefit outweighs the potential risk. Patients should be cautioned to avoid getting the drug powder into their eyes. They should be advised that this may result in precipitation or worsening of narrow-angle glaucoma, eye pain or discomfort, temporary blurring of vision, visual halos or coloured images in association with red eyes from conjunctival congestion and corneal oedema. Should any combination of these eye symptoms develop, patients should stop using tiotropium bromide and consult a specialist immediately. Tiotropium bromide should not be used more frequently than once a day. Spiriva capsules contain 5.5mg lactose monohydrate. Interactions: Although no formal drug interaction studies have been performed, tiotropium bromide inhalation powder has been used concomitantly with other drugs without clinical evidence of drug interactions. These include sympathomimetic bronchodilators, methylxanthines, oral and inhaled steroids, commonly used in the treatment of COPD. The co-administration of tiotropium bromide with other anticholinergic-containing drugs has not been studied and is therefore not recommended. Pregnancy and Lactation: No documented clinical data on exposed pregnancies are available. The potential risk for humans is unknown. Tiotropium bromide should therefore only be used during pregnancy when clearly indicated. It is unknown whether tiotropium bromide is excreted in human breast milk. Use of tiotropium bromide during breast feeding is not recommended. A decision on whether to continue or discontinue breast feeding or therapy with tiotropium bromide should be made taking into account the benefit of breast feeding to the child and the benefit of tiotropium bromide therapy to the woman. Effects on ability to drive and use machines: No studies have been performed. The occurrence of dizziness, blurred vision, or headache may influence the ability to drive and use machinery. Undesirable effects: Common ($\geq 1/100$ to $<1/10$) Dry mouth. Uncommon ($\geq 1/1000$ to $<1/100$) Dizziness, headache, taste disorders, vision blurred, atrial fibrillation, pharyngitis, dysphonia, cough, stomatitis, gastrooesophageal reflux disease, constipation, nausea, rash, dysuria, urinary retention. Events of unknown frequency not attributed to tiotropium in clinical trials but considered to be adverse drug reactions: dehydration, dental caries, angioneurotic oedema, skin infection, skin ulcer, dry skin, joint swelling. Serious undesirable effects consistent with anticholinergic effects include glaucoma, constipation and intestinal obstruction including ileus paralytic as well as urinary retention. An increase in anticholinergic effects may occur with increasing age. Prescribers should consult the Summary of Product Characteristics for further information on side effects. Pack sizes and NHS price: Combopack HandiHaler device and 30 capsules (3 blister strips) £34.87 Refill Pack 30 capsules (3 blister strips) £31.89. Legal category: POM Marketing Authorisation Holder: Boehringer Ingelheim International GmbH, D-55216 Ingelheim am Rhein, Germany. MA Numbers: PL 14598/0062. Prescribers should consult the Summary of Product Characteristics for full prescribing information. Prepared in February 2010.

Adverse events should be reported. Reporting forms and information can be found at www.yellowcard.gov.uk. Adverse events should also be reported to Boehringer Ingelheim Drug Safety on 0800 328 1627 (freephone).

Spiriva has been developed by Boehringer Ingelheim and is being co-promoted by Pfizer Limited and Boehringer Ingelheim Limited.

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