

David MG Halpin

Consultant Physician, Royal Devon & Exeter Hospital, barrack Road, Exeter EX2 5DW, UK

Diagnosing COPD

It is widely recognized that many people with chronic obstructive pulmonary disease (COPD) are undiagnosed, including some with significant airflow obstruction. The best available data suggest that the prevalence of physiologically defined COPD in adults aged 40 years is 9%–10% (Halbert et al 2006). In their study of randomly selected individuals aged 60 to 74 years from a UK general practice register, Dickinson and colleagues (1999) found that there were 2.69 “true” cases of COPD for each diagnosed case, with an overall prevalence of 6.2% with undiagnosed COPD. A similar study of a random sample of the general population aged over 64 years in Finland found 1.99 and 1.62 true cases of COPD for each diagnosed case in men and women, respectively (Isoha et al 1994). Other studies have compared the prevalence of respiratory symptoms with patient-reported diagnoses and found a ratio of true COPD cases to self-reported diagnoses ranging from approximately 1.5 to 4 (Littlejohns et al 1989; Lundback et al 1991; Viegi et al 1991; Lai et al 1995). Most recently a study in Manchester by Frank and colleagues (2006) found that nearly two thirds of patients with spirometrically confirmed COPD had no prior diagnosis of COPD and nearly half of these had severe airflow obstruction.

Some of these patients have no diagnosis of airways disease at all and others are misdiagnosed as asthmatics. It has been suggested that COPD and asthma are simply different phenotypes of a common obstructive disease; the so called Dutch Hypothesis (Bleecker 2004). This has led to the suggestion that differentiation is unnecessary, particularly as treatment uses similar drugs (Kraft 2006). However, most evidence suggests that asthma and COPD have a different etiology and pathophysiology, and require different management (Barnes 2006). Although similar drugs are used in both diseases they are used in different sequences and with different doses. For example, low dose inhaled steroids are essential in patients with asthma whose symptoms are persistent, whilst in COPD inhaled steroids are principally recommended in patients with an forced expiratory volume in one second (FEV_1) of <50% and experiencing frequent exacerbations. The evidence for their efficacy is based on use at a high dose (Fabbri and Hurd 2003; NICE 2004).

Perhaps the most important reason for distinguishing the two conditions is the fact that they have very different prognoses. Most asthmatics maintain normal or near normal lung function and have a normal life expectancy (McFadden 2000) whereas lung function in patients with COPD declines inexorably and patients have a reduced life expectancy (Soriano et al 2000).

Making a diagnosis of COPD and its differentiation from asthma presents an important challenge for primary care practitioners. Previously, many practitioners used reversibility testing as a sensitive and specific test to differentiate COPD from asthma. There is now clear evidence that, unless interpreted in the light of the clinical assessment of the diagnosis, reversibility testing alone cannot differentiate asthma from COPD. The results are not reproducible over time (Calverley et al 2003) and are highly dependent on the method used to assess reversibility (Nisar et al 1990; Hadcroft and Calverley 2001). Some degree of fixed airflow obstruction occurs in up to 30% of patients with asthma (Kesten and Rebuck 1994) whilst many patients with COPD show a significant bronchodilator response despite having no clinical features of asthma (Tashkin and Celli 2005). Moreover, if a fixed threshold such as

a 15% improvement in FEV₁ is used reversibility testing has been found to have a sensitivity of only 44% for detecting asthma and a specificity of only 72% for distinguishing asthma from COPD (Mannino et al 2000).

It has been pointed out that the recommendation in the National Institute for Clinical Excellence guideline that routine reversibility testing should not be relied upon to differentiate COPD from asthma (NICE 2004) makes it more difficult for primary care practitioners to diagnose COPD. Prior to this recommendation, some clinicians preferred to use a flawed test to make a wrong diagnosis easily rather than putting the effort in to making a correct diagnosis (Frank et al 2006).

When making a diagnosis, thinking of possible diagnoses is the first and possibly the most important step. If a diagnosis is not considered then subsequent history taking, examination, and investigations will be markedly less likely to lead to that diagnosis. The results of the CADRE (COPD and Asthma Diagnostic/Management Reassessment) study reported by Pearson and colleagues (2006) in this edition of International Journal of COPD highlights this issue. The authors found that a working diagnosis of asthma was more likely to be made in younger patients, women, and nonsmokers. The authors found that it was much more common to revise the diagnosis of asthma than to revise the diagnosis of COPD after clinical review and spirometry.

What are the implications for practice? The CADRE study supports the need to take a good clinical history when making a diagnosis and to use spirometry to confirm the diagnosis. Since this work was undertaken, the availability of spirometry in primary care has increased considerably in the UK (Halpin and Connellan 2005) and its routine use has been encouraged by contractual arrangements with general practitioners. The CADRE study also reminds us that COPD is becoming more common in women and suggests that there was a tendency to over-diagnose asthma and under-diagnose COPD in primary care. Reluctance to make the diagnosis of COPD and gender bias when diagnosing COPD are well recognized (Chapman et al 2001) and these must be overcome if the missing millions with COPD are to be identified.

References

- Barnes PJ. 2006. Against the Dutch hypothesis: asthma and chronic obstructive pulmonary disease are distinct diseases. *Am J Respir Crit Care Med*, 174:240-3; discussion 243-4.
- Bleecker ER. 2004. Similarities and differences in asthma and COPD: The Dutch hypothesis. *Chest*, 126(2 Suppl):93S-5S.
- Calverley PM, Burge PS, Spencer S, et al. 2003. Bronchodilator reversibility testing in chronic obstructive pulmonary disease. *Thorax*, 58:659-64.
- Chapman KR, Tashkin DP, Pye DJ. 2001. Gender bias in the diagnosis of COPD. *Chest*, 119:1691-5.
- Dickinson JA, Meaker M, Searle M, et al. 1999. Screening older patients for obstructive airways disease in a semi-rural practice. *Thorax*, 54:501-5.
- Fabbri LM, Hurd SS. 2003. Global strategy for the diagnosis, management and prevention of COPD: 2003 update. *Eur Respir J*, 22:1-2.
- Frank TL, Hazell ML, Linehan MF, et al. 2006. The diagnostic accuracies of chronic obstructive pulmonary disease (COPD) in general practice: the results of the MAGIC (Manchester Airways Group Identifying COPD) study. *Prim Care Respir J*, 15:286-93.
- Hadcroft J, Calverley PM. 2001. Alternative methods for assessing bronchodilator reversibility in chronic obstructive pulmonary disease. *Thorax*, 56:713-20.
- Halbert RJ, Natoli JL, Gano A, et al. 2006. Global burden of COPD: systematic review and meta-analysis. *Eur Respir J*, 28:523-32.
- Halpin DMG, Connellan S. 2005. Confidence and understanding among general practitioners and practice nurses about diagnosis and management of chronic obstructive pulmonary disease. *Thorax*, 60(suppl 2):ii95.
- Isoaho R, Puolijoki H, Huhti E, et al. 1994. Prevalence of chronic obstructive pulmonary disease in elderly Finns. *Respir Med*, 88:571-80.
- Kesten S, Rebuck AS. 1994. Is the short-term response to inhaled beta-adrenergic agonist sensitive or specific for distinguishing between asthma and COPD? *Chest*, 105:1042-5.
- Kraft M. 2006. Asthma and chronic obstructive pulmonary disease exhibit common origins in any country! *Am J Respir Crit Care Med*, 174:238-40; discussion 243-4.
- Lai CK, Ho SC, Lau J, et al. 1995. Respiratory symptoms in elderly Chinese living in Hong Kong. *Eur Respir J*, 8:2055-61.
- Littlejohns P, Ebrahim S, Anderson R. 1989. Treatment of adult asthma: is the diagnosis relevant? *Thorax*, 44:797-802.
- Lundback B, Nystrom L, Rosenhall L, et al. 1991. Obstructive lung disease in northern Sweden: respiratory symptoms assessed in a postal survey. *Eur Respir J*, 4:257-66.
- Mannino DM, Gagnon RC, Petty TL, et al. 2000. Obstructive lung disease and low lung function in adults in the United States: data from the National Health and Nutrition Examination Survey, 1988-1994. *Arch Intern Med*, 160:1683-9.
- McFadden ER, Jr. 2000. Natural history of chronic asthma and its long-term effects on pulmonary function. *J Allergy Clin Immunol*, 105(2 Pt 2):S535-9.
- [NICE] National Institute for Clinical Excellence (NICE). 2004. Chronic obstructive pulmonary disease. National clinical guideline for management of chronic obstructive pulmonary disease in adults in primary and secondary care. *Thorax*, 59(Suppl 1):1-232.
- Nisar M, Walshaw M, Earis JE, et al. 1990. Assessment of reversibility of airway obstruction in patients with chronic obstructive airways disease. *Thorax*, 45:190-4.
- Pearson M, Ayres JG, Sarno M, et al. 2006. Diagnosis of airway obstruction in primary care in the UK: the CADRE (COPD and Asthma Diagnostic/management REassessment) programme 1997-2001. *Int J COPD*, 1:435-43.
- Soriano JB, Maier WC, Egger P, et al. 2000. Recent trends in physician diagnosed COPD in women and men in the UK. *Thorax*, 55:789-94.
- Tashkin D, Celli B, Decramer M, et al. 2005. Bronchodilator responsiveness in COPD patients enrolled in the UPLIFT trial. *Proc Am Thorac Soc*, 2(Abstract):A656.
- Viegi G, Paoletti P, Carrozzi L, et al. 1991. Prevalence rates of respiratory symptoms in Italian general population samples exposed to different levels of air pollution. *Environ Health Perspect*, 94:95-9.