COPD: Definition, epidemiology and diagnosis

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Introduction

Chronic obstructive pulmonary disease (COPD) is rapidly becoming the focus of health strategies and management guidelines as planners recognise the increasing impact this disease will have on health services. As the epidemiology of COPD mirrors the epidemiology of tobacco smoking we can expect an increasing burden of COPD in our society, particularly in women, for the next decade and beyond. This paper covers essential terminology, epidemiology, and diagnosis.

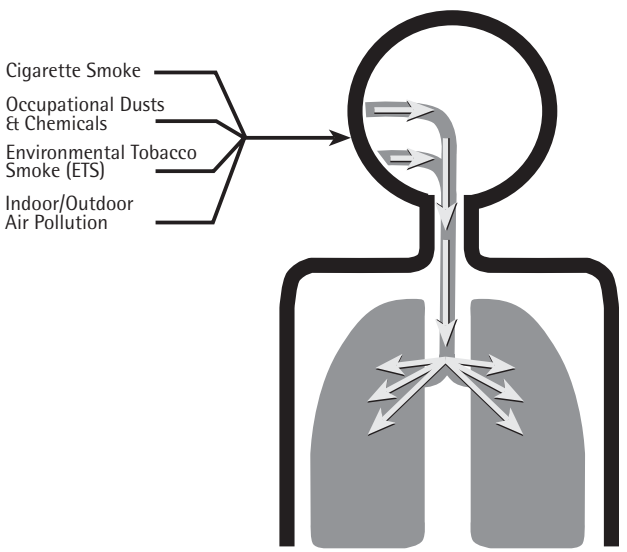
Definitions and terminology

COPD is not a term that either doctors or their patients understand intuitively. After a painstaking process of consultation, the international consortium represented by the acronym GOLD (Global initiative for chronic Obstructive Lung Disease) settled on this term.[1] We should support this decision because for many years there was an increasingly confusing set of terms used which detracted from patients and doctors gaining an understanding of the condition.

In the past, the terms 'emphysema' and 'chronic bronchitis' were frequently used. Emphysema is a pathological term only really applicable when there is CT scan or post-mortem evidence of destruction of alveolar tissue. Chronic bronchitis was defined by the Medical Research Council (UK) as the presence of cough and sputum for at least three months in each of two consecutive years. In practice this definition was rarely helpful and does not adequately emphasise the extent to which airflow obstruction is a characteristic feature. The definition, which has been adopted by the GOLD Working Party, is as follows:

COPD is the 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.[1]

*Figure 1. Total Burden of Inhaled Particles**

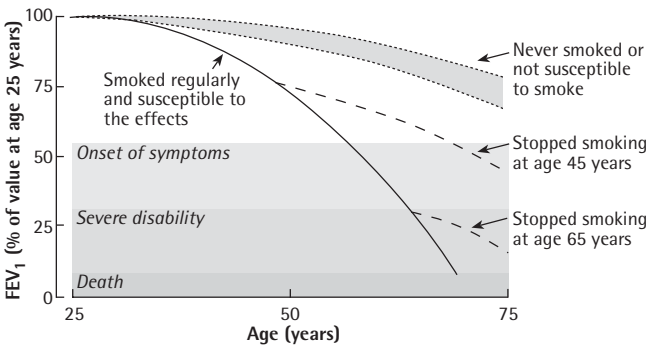


* *Global Initiative for Chronic Obstructive Lung Disease. Reproduced with permission .*

Prevalence

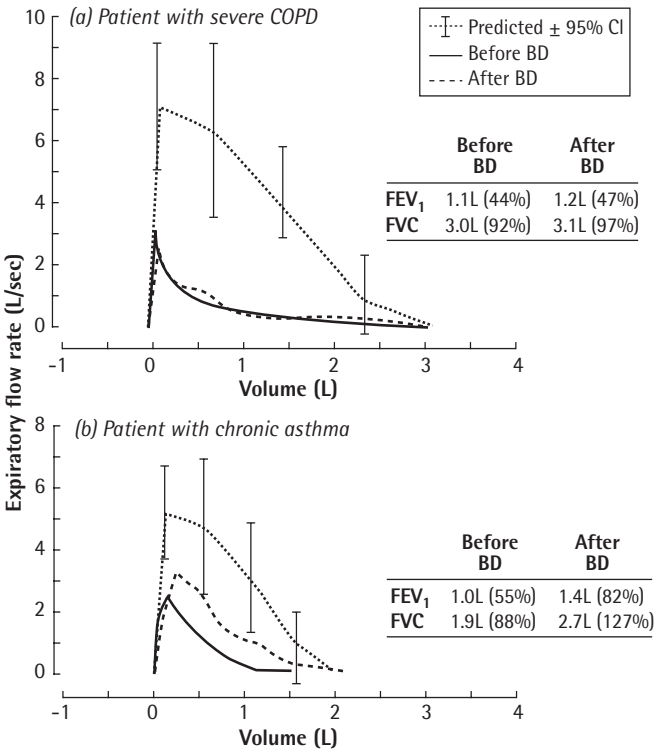
In New Zealand we have no accurate prevalence data for COPD. Overseas estimates from similar western countries such as the UK suggest a prevalence of around 6% of the total population.[2] As the diagnosis rests on the confirmation of airflow obstruction by spirometry there is the potential for mis-diagnosis.

*Figure 2. Time-course of chronic obstructive pulmonary disease (COPD)**



The figure shows the rate of loss of forced expiratory flow in one second (FEV_1) for a hypothetical susceptible smoker, and the potential effect of stopping smoking early or late in the course of COPD. Other susceptible smokers will have different rates of loss, thus reaching 'disability' at different ages. The normal FEV_1 ranges from below 80% to above 120%, so this will affect the starting point for the individual's data (not shown).

*Figure 3. Maximal expiratory flow-volume curves in severe chronic obstructive pulmonary disease (COPD) and chronic asthma**



The patient with COPD has reduced peak expiratory flow, and severely decreased flows at 25%, 50% and 75% of vital capacity compared with the normal range (vertical bars), and shows minimal response to bronchodilator (BD). By comparison, the patient with chronic asthma shows incomplete, but substantial, reversibility of expiratory flow limitation across the range of vital capacity. After BD the forced expiratory volume in one second (FEV_1) was within the normal range (82% predicted). Absolute and per cent predicted values for FEV_1 and forced vital capacity (FVC) before and after BD are shown for each patient.

* *Figures 2 and 3 – The COPDX plan: Australian and New Zealand guidelines for the management of chronic obstructive pulmonary disease 2003. MJA 2003; 178 Supplement:–.*

Pathogenesis

The definition recognises the increasing emphasis on inflammation in our understanding of the development and progression of airflow limitation. Although it has been known for many years that the main risk factor for the development of COPD is tobacco smoking, it is now clear that other noxious particles and gases can add to the pulmonary burden and have their impact through the aggravation of inflammation in small airways (Figure 1). In New Zealand we do have some data with regard to occupational risk and higher risk occupations include bakers, food processors, spray painters, and chemical processors.[3] In a small percentage of cases a deficiency of alpha-1 antitrypsin is the predisposing factor and in those who smoke, early onset of COPD may occur in the 3rd or 4th decade.

Natural history

Unfortunately the development of airflow obstruction and the attendant symptoms occur insidiously and this often leads to a delay in recognising the diagnosis. Although the natural history can be variable in individual patients, overall there is a progressive course and this is aggravated if the exposure to tobacco smoke or other occupational dusts is continued (Figure 2).

Making the diagnosis

The diagnosis should be made in primary care and ideally it should be made by proactively screening all registered patients who are either tobacco smokers, working in at-risk occupations or who present with symptoms suggestive of COPD.

The main symptoms are chronic cough and sputum production and exertional breathlessness. In the latter stages of disease patients are more likely to present during an acute exacerbation, which is generally provoked by a respiratory infection – viral or bacterial – and may lead to an aggravation of symptoms or the development of complications such

as pneumonia, pneumothorax or right heart failure.

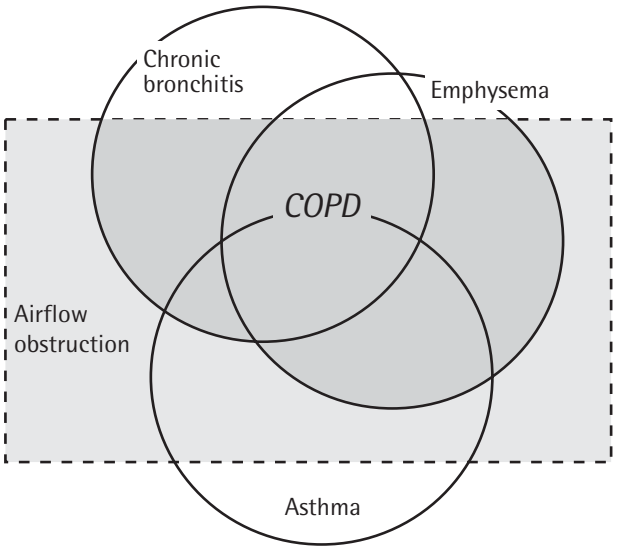
Confirming the diagnosis rests on the availability of pulmonary function testing (Figure 3). Office spirometry is now more widely available in New Zealand and, providing the instrument is correctly calibrated and the interpretation sound, this is a perfectly acceptable means of diagnosis. The key element is identifying the presence of airflow obstruction (FEV_1/VC ratio of less that 70%). Alternatively, pulmonary function testing with appropriate reporting can be readily obtained in pulmonary function laboratories in most centres in New Zealand.[4] Most laboratories will offer reversibility testing with a bronchodilator such as salbutamol.

Differential diagnosis

The most important condition to exclude in assessing patients with airflow obstruction and appropriate symptoms is asthma. If the airflow limitation is fully or substantially reversible (>15% improvement in FEV_1) then the patient should be considered as having asthma (Figure 4). Other conditions which may present with similar symptoms are shown in Tables 1 and 2.

The key differentiating features for COPD is the history of 20+ pack

*Figure 4. Overlap of bronchitis, emphysema and asthma within chronic obstructive pulmonary disease (COPD)**



This non-proportional Venn diagram shows he overlap of chronic bronchitis, emphysema and asthma within COPD. Chronic bronchitis, airway narrowing and emphysema are independent effects of cigarette smoking, and may occur in various combinations. Asthma is, by definition, associated with reversible airflow obstruction. Patients with asthma whose airflow obstruction is completely reversible do not have COPD. In many cases it is impossible to differentiate patients with asthma whose airflow obstruction does not remit completely from persons with chronic bronchitis and emphysema who have partially reversible airflow obstruction with airway hyperreactivity.

* *The COPDX plan: Australian and New Zealand guidelines for the management of chronic obstructive pulmonary disease 2003. MJA 2003; 178 Supplement: –. ©Copyright 2003. The Medical Journal of Australia. Reproduced with permission.*

Table 1. Differential diagnosis of COPD

There are many conditions that can cause respiratory symptoms similar to those of COPD. These include (in approximate order of frequency of occurrence):

1. Upper Respiratory Tract (URT) disease (e.g. sinusitis) causing post nasal drip and coughing
2. Post infective bronchial hyper-responsiveness, including whooping cough (can only be definitively diagnosed if tested for with a bronchial challenge)
3. Lung cancer obstructing an airway
4. Left ventricular failure
5. Vocal cord dysfunction
6. Hyperventilation
7. Bronchiectasis

Table 2. Differences between asthma and chronic obstructive pulmonary disease

Feature	Asthma	COPD
Age of onset	Often younger (childhood)	Usually older
Rapid onset	Often	Rare
Smoking history	Sometimes	Almost always
Allergic background	Often	Seldom
Cough sputum	Sometimes	Often
Wheeze	Often	Sometimes
Diurnal peak flow variability	Usual	Sometimes
Bronchial hyper-responsiveness†	Usual	Sometimes
Reversible airflow obstruction‡	Usual	Sometimes

† Defined as a $PC_{20} \leq 8$ mg/ml methacholine or histamine.

‡ Defined as a 15% or greater improvement in FEV_1 15 minutes after 400 µg inhaled salbutamol.

*Figure 5. Classification of severity of chronic obstructive pulmonary disease (COPD)**

	COPD severity		
Factor	**Mild**	**Moderate**	**Severe**
Spirometry findings – postbrochodilator FEV_1	60%–80% predicted	40%–59% predicted	<40% predicted
Functional assessment (activities of daily living)	Few symptoms No effects on daily activities Breathless on moderate exertion	Increasing dyspnoea Breathless on the flat Increasing limitation of daily activities	Dyspnoea on minimal exertion Daily activities severely curtailed
Complications	No	Exclude complications; consider sleep apnoea if there is pulmonary hypertention	Severe hypoxaemia (PaO_2 <60mmHg, or 8kPa) Hypercapnia ($PaCO_2$ >45 mmHg, or 6kPa) Pulmonary hypertension Heart failure Polycythaemia

FEV_1 = forced expiratory volume in one second.
PaO_2 = partial pressure of oxygen, arterial. $PaCO_2$ = partial pressure of carbon dioxide, arterial.

* *The COPDX plan: Australian and New Zealand guidelines for the management of chronic obstructive pulmonary disease 2003. MJA 2003; 178 Supplement:–.*

years of tobacco smoking and in asthma the association with atopy, a positive family history and the earlier age of onset.

Other conditions which need to be excluded from COPD in an older patient presenting with breathlessness include left ventricular failure, pulmonary embolism, interstitial lung disease or the insidious development of a pleural effusion. In this regard, in addition to spirometry, the single most useful baseline investigation is a chest radiograph which will assist in identifying other obvious pulmonary conditions.

Classification of severity

Figure 5 shows the recommended classification of severity adopted by the TSANZ Working Party.[2] This classification is based on the absolute value of the FEV_1 compared to predicted values. Mild COPD is defined as a FEV_1 of greater than 60% of predicted (with an impaired ratio), moderate an FEV_1 of 40–59% of predicted, and severe an FEV_1 of 40% of predicted or below. In this latter group arterial hypoxaemia is often a feature and screening for this (SaO_2 less than 92%) with pulse oximetry is recommended. Arterial blood gas analysis is required for those with significant hypoxaemia to determine whether domiciliary oxygen may be appropriate and to assess the degree, if any, of hypercapnia.

In addition to measuring the FEV_1 it is important to quantify the degree of dyspnoea. Traditionally this has not been the focus of much attention, which makes it difficult when judging the effect of treatment or documenting changes over time. The recommended grading system is that developed by the Medical Research Council and this is shown in Table 3.

Websites which contain useful information with regard to COPD include:

1. GOLD website: www.goldcopd.com
2. The Australian Lung Foundation: www.lungnet.org.au
3. The Asthma & Respiratory Foundation of New Zealand: www.asthmanz.co.nz

Table 3. Medical Research Council grading of functional limitation due to dyspnoea

Grade	Symptom complex
1	'I only get breathless with strenuous exercise.'
2	'I get short of breath when hurrying on the level or walking up a slight hill.'
3	'I walk slower than most people of the same age on the level because of breathlessness or have to stop for breath when walking at my own pace on the level.'
4	'I stop for breath after walking about 100 metres or after a few minutes on the level.'
5	'I am too breathless to leave the house' or 'I am breathless when dressing.'

References

1. NIH. Global initiative for chronic obstructive lung disease. National Heart, Lung and Blood Institute. Publication Number 2701. April 2001.
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3. Fishwick D, Bradshaw LM, d'Souza W et al. Chronic bronchitis, shortness of breath and airway obstruction by occupation in New Zealand. Am J Respir Crit Care Med 1997; 156:1440–6.
4. Johns DP and Pierce R. McGraw-Hill's pocket guide to spirometry. McGraw-Hill. In press 2003.