CORRESPONDENCE

GOLD COPD classification and prognostic pessimism regarding ICU admission

Incorporation of the Global Initiative for Chronic Obstructive Lung Disease (GOLD) classification of severity of expiratory airflow limitation in chronic obstructive pulmonary disease (COPD) into the recent National Institute for Health and Clinical Excellence (NICE) guidelines is welcome and sensible. Describing a forced expiratory volume in one second (FEV\textsubscript{1}) of 51% predicted as ‘mild disease’ fails to capture the loss of lung function and irreversible damage done. Recognition and optimal early management of COPD cannot be overemphasised to limit its long-term health consequences.

However, we have concerns that its adoption without adequate explanation in the UK could have unintended negative consequences in this patient group if presenting acutely unwell, when decisions regarding intensive care and use of invasive mechanical ventilation (IMV) are being made. Widely varying ICU admission criteria and prognostic pessimism among UK critical care physicians regarding COPD have been demonstrated. The description of a condition as ‘severe’, which could include those with an FEV\textsubscript{1} of up to 50% predicted and is not a comment on general functional capacity or physical frailty, may be misinterpreted by clinicians. This could then contribute to an overly nihilistic view of potential outcome and hence inappropriate refusal of intensive care for some who could benefit.

The recent National Chronic Obstructive Pulmonary Disease Resources and Outcomes Project report concerning acidosis and use of non-invasive ventilation (NIV) in COPD highlights several important issues regarding acute care. The use of IMV was low; 110 out of 2143 acidosic patients received IMV and only 54 out of 1077 patients receiving NIV had treatment escalated to IMV. Given the methodology of this survey, it must be considered representative of UK practice.

First, we would suggest that in addition to explaining the reclassification and its meaning to patients as O’Reilly and Rudolf suggest, this change needs to be shared with colleagues responsible for acutely ill COPD patients. Second, care should be taken with clinical letters and discharge documentation. Many hospitals have now adopted electronic patient record systems enabling clinical letters to be viewed without the paper notes being present. We would suggest that in addition to the GOLD classification, functional exercise capacity is recorded besides the absolute and predicted values of FEV\textsubscript{1} and forced vital capacity.

By being aware of potential problems, we can hopefully gain the benefits of bringing our practice in line with international colleagues without disadvantaging a vulnerable group.

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Competing interests None.

Provenance and peer review Not commissioned; internally peer reviewed.

Accepted 16 March 2011
Published Online First 17 April 2011
Thorax 2012;67:263.
doi:10.1136/thoraxjnl-2011-200134

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Authors’ response: ‘What’s nice about the new NICE guideline?’

We thank the correspondents for these kind and helpful comments. In adopting the Global Initiative on Obstructive Lung Disease (GOLD) classification of severity of airflow obstruction, the National Institute for Health and Clinical Excellence (NICE) guideline update has introduced consistency with international guidelines including those of the American Thoracic Society and the European Respiratory Society. The NICE guidelines note that this classification relates specifically to degrees of airflow obstruction which are arbitrary and may not be closely related to degrees of clinical severity in chronic obstructive pulmonary disease (COPD). The current use of the term ‘severe’ for airflow obstruction with forced expiratory volume in 1 s (FEV\textsubscript{1})<50% in place of ‘moderate’ (NICE 2004) may also help to underline the potentially serious nature of the lung function impairment and encourage smoking cessation and more active management.

The NICE guidelines stress the overriding importance of clinical criteria to assess COPD severity, and promote multidimensional assessment using a range of tools to assess breathlessness and functional capacity, ranging from the simple Medical Research Council (MRC) scale to the BODE Index, which includes breathlessness, BMI and exercise capacity as well as lung function. Outcomes in COPD are known to be related to clinical factors, including severity of symptoms and exacerbation frequency, as well as lung function. These should be taken into account, together with comorbidities, in assessing patients admitted to hospital with acute exacerbation of COPD and in whom intensive care and use of mechanical ventilation is to be considered. It is acknowledged that there is variation in intensive care unit criteria for admission to manage COPD. This suggests a need for clear evidence-based criteria for intensive care support and intermittent mandatory ventilation (IMV) based on valid prognostic indicators rather than on a diagnostic classification of severity of airflow obstruction which is not intended for this purpose. Evidence-based guidance for the use of non-invasive ventilation (NIV) uses criteria other than severity of airflow obstruction. Failure of NIV leading to the need for IMV is predicted not by lung function but by the Acute Physiology and Chronic Health Evaluation II (APACHE II) score, pH, respiratory rate, and Glasgow coma score.

The authors acknowledge the National COPD Resources and Outcome Project (NCROP) evidence of low use of IMV in patients with COPD, and agree that the data suggest a variable degree of nihilism for which there is no clear justification. The NICE guidelines note that the decision on which patients with exacerbations of COPD will benefit from intubation is difficult, and involves balancing health status with an estimate of expectation of survival. Factors that are likely to influence this decision are prior functional status, BMI requirement for oxygen when stable, comorbidities and previous intensive treatment unit (ITU) admissions. Physiological thresholds for use of IMV have not been subjected to systematic evaluation and decisions are currently based on clinical judgement rather than objective data. The severity of the acute illness (APACHE II), associated comorbidity and malignancy are predictors of in-hospital mortality in patients with COPD and acute respiratory failure. There is clearly a need for further evidence-based assessment of predictors of outcome from IMV rather than inappropriate reliance on diagnostic stratification of FEV\textsubscript{1}.

The authors agree that there is a need to explain the reclassification and its meaning.
Importance of past occupational exposures in the rising incidence of idiopathic pulmonary fibrosis in the UK

We read with interest the recent article by Navaratnam et al highlighting the unexplained rising incidence of idiopathic pulmonary fibrosis (IPF) in the UK. While we agree that this area is of great clinical interest, we feel that the rapidly rising incidence, linked with the gender, age, geographical and socioeconomic risk factors for this disease, is strongly suggestive that the cause is not wholly idiopathic. Previous work by the same group found that 20% of IPF could be explained epidemiologically by occupational exposures to metals or wood dust, yet there is no discussion relating to how these or other exposures may have changed over the time period studied.

Mortality due to asbestosis is also likely to be highly relevant here, and in a separate paper published recently, the same research group has reported a 10-fold rise in asbestosis mortality from death certificate data (13 in 1968 to 129 in 2006). The authors went on to note that the rising asbestosis mortality mirrors the rising trend in mesothelioma mortality, where over a similar period deaths rose from 135 to 2085.

Given that the mortality from IPF clinical syndrome seems to be rising in parallel to that of mesothelioma, and that these diseases have similar demographic risk factors, the obvious question that arises is how much IPF is actually due to asbestosis exposure that has not been recognised in life, or not recorded on the death certificate? Asbestosis usage in UK industry was widespread up to the 1980s, as demonstrated by a recent mesothelioma study where two-thirds of the randomly selected male controls born in the 1940s were found to have worked in at least one high or medium risk job for asbestos exposure. We agree that this area is of great clinical interest, we feel that the rapidly rising incidence, linked with the gender, age, geographical and socioeconomic risk factors for this disease, is strongly suggestive that the cause is not wholly idiopathic. Previous work by the same group found that 20% of IPF could be explained epidemiologically by occupational exposures to metals or wood dust, yet there is no discussion relating to how these or other exposures may have changed over the time period studied.

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These data suggest that it is likely that a large proportion of UK males presenting with pulmonary fibrosis aged 60–70 years will have previously been occupationally exposed to asbestos in the 1950s–1980s, whether or not they report it when questioned 40–50 years later. This is compounded by the similar radiological features shared by asbestosis and usual interstitial pneumonitis, as well as the problems of interpreting asbestosis fibre counts if available. These diagnostic difficulties, linked with the known inaccuracies of death certificate data, and no understanding of individual susceptibility are likely to make establishing a clear epidemiological link between IPF and asbestos challenging.

While we agree with the authors that more research is required in this fascinating area, we believe the term ’idiopathic’ may be misleading, and that a significant proportion of UK IPF is likely to relate to past occupational exposures. Given that UK peak asbestos usage was in the late 1960s, and with reference to a US model, mortality from asbestosis should peak around the same time as that of mesothelioma, sometime between now and 2020. The relative change in mortality from mesothelioma, asbestosis and IPF over the next decade will therefore be of great interest, and may give us a valuable insight into the true relationship between asbestos exposure and pulmonary fibrosis.

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Competing interests None.

Authors’ response
We thank Drs Barber and Fishwick for their interest in our paper and would like to briefly respond to their comments. Our paper describes a pragmatic epidemiological study with individuals who had an underlying diagnosis of idiopathic pulmonary fibrosis clinical syndrome (IPF-CS). The aim was to investigate recent temporal trends in incidence and survival and not to consider environmental exposures that may have an aetiological link. Hence, although this increase may be linked to occupational or other environmental exposures, our data do not permit firm conclusions to be drawn. It is also possible that the rapid increase in incidence reported is due to an increasing tendency to investigate patients in the UK.