to patients and colleagues responsible for acutely ill patients with COPD. In keeping with the NICE guidelines, COPD severity should be described in terms of functional status using at least the modified MRC score, as well as previous severity of lung function impairment. It should be made clear that the severity of lung function impairment in COPD does not necessarily correlate with clinical severity or outcome of inpatient care, which may include ITU and IMV management.

John O'Reilly,¹ Michael Rudolf²

¹Aintree University Hospital NHS Trust, Liverpool, UK;

²Ealing Hospital NHS Trust, London, UK

Correspondence to John O'Reilly, Consultant Physician, Aintree University Hospital NHS Trust, Lower Lane, Liverpool L9 7AL, UK; john.oreilly@aintree.nhs.uk

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; internally peer reviewed.

Accepted 16 March 2011

Published Online First 17 April 2011

Thorax 2012;**67**:263–264.

doi:10.1136/thoraxjnl-2011-200181

REFERENCES

1. Collins A, Walden A, Parker R. GOLD COPD classification and prognostic pessimism regarding ICU admission. *Thorax* 2012;**67**:263.
2. National Institute for Health and Clinical Excellence. *Chronic obstructive pulmonary disease. NICE clinical guideline (update) consultation*. London: National Institute for Health and Clinical Excellence, 2010.
3. Celli BR, Cote CG, Marin JM, et al. The body-mass index, airflow obstruction, dyspnea, and exercise capacity index in chronic obstructive pulmonary disease. *N Engl J Med* 2004;**350**:1005–12.
4. Puhon M, Garcia-Aymerich J, Frey M, et al. Expansion of the prognostic assessment of patients with chronic obstructive pulmonary disease: the updated BODE index and the ADO index. *Lancet* 2009;**374**:704–11.
5. Jones RC, Donaldson GC, Chavannes NH, et al. Derivation and validation of a composite index of severity of chronic obstructive pulmonary disease: the DOSE index. *Am J Respir Crit Care Med* 2009;**180**:1189–95.
6. Pickard AS, Jung E, Bartle B, et al. Coded cause of death and timing of COPD diagnosis. *COPD* 2009;**6**:41–7.
7. Lightowler JV, Wedzicha JA, Elliot MW, et al. NIPPV to treat respiratory failure resulting from acute exacerbation of COPD: Cochrane systematic review and meta analysis. *BMJ* 2003;**326**:185.
8. Confalonieri M, Garuti G, Cattaruzza MS, et al. A chart of failure risk for noninvasive ventilation in patients with COPD exacerbation. *Eur Respir J* 2005;**25**:348–55.
9. Köhlein T, Welte T. *Ventilation in obstructive lung disease respiratory emergencies*. Vol. 36. European Respiratory Society Monograph, 2006:34–48.
10. Nevins ML, Epstein SK. Predictors of outcome for patients with COPD requiring invasive mechanical ventilation. *Chest* 2001;**119**:1840–9.

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.¹ 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,² 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).³ 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 2058.

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 asbestos exposure that has not been recognised in life, or not recorded on the death certificate? Asbestos 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.⁴

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 asbestos 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 occupa-

tional exposures. Given that UK peak asbestos usage was in the late 1960s, and with reference to a US model,⁵ 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.

C M Barber, D Fishwick

Centre for Workplace Health, Royal Hallamshire Hospital, Sheffield, UK

Correspondence to Dr Christopher Michael Barber, Centre for Workplace Health, Royal Hallamshire Hospital, Sheffield S10 2JF, UK; chris.barber2@sth.nhs.uk

Competing interests None.

Contributors This letter is co-written by both authors.

Provenance and peer review Not commissioned; internally peer reviewed.

Accepted 9 August 2011

Published Online First 7 September 2011

Thorax 2012;**67**:264.

doi:10.1136/thoraxjnl-2011-200836

REFERENCES

1. Navaratnam V, Fleming KM, West J, et al. The rising incidence of idiopathic pulmonary fibrosis in the UK. *Thorax* 2011;**66**:462–7.
2. Hubbard R, Lewis S, Richards K, et al. Occupational exposure to metal or wood dust and aetiology of cryptogenic fibrosing alveolitis. *Lancet* 1996;**347**:284–9.
3. Hanley A, Hubbard RB, Navaratnam V. Mortality trends in asbestosis, extrinsic allergic alveolitis and sarcoidosis in England and Wales. *Respir Med* 2011;**105**:1373–9.
4. Rake C, Gilham C, Hatch J, et al. Occupational, domestic and environmental mesothelioma risks in the British population: a case-control study. *Br J Cancer* 2009;**100**:1175–83.
5. Dos Santos Antao VC, Pinheiro GA, Wassell JT. Asbestosis mortality in the USA: facts and predictions. *Occup Environ Med* 2009;**66**:335–8.

Authors' response

We thank Drs Barber and Fishwick for their interest in our paper and would like to briefly respond to their comments.¹ Our paper describes a pragmatic epidemiological study with individuals who had an underlying diagnosis of idiopathic pulmonary fibrosis clinical syndrome (IPF-CS).² 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.