we have conflict of interest statements, which can hide a multitude of sins. Should we apply the News of the World test—if what we are NOW doing was on the front page of that newspaper, how would we feel? Big Pharma—let's have all data on all patients readily available on line, and make sure recent lawsuits over concealed data never happen again. Hospital Trusts—be open if there are problems such as surgical mortality, and be open about how they will be dealt with. The other side of the coin—let us all be adults, accept that human beings are human and make mistakes despite everything, and not

degenerate into a flood tide of frothing indignation if something has gone wrong (certain sections of the media take note). 'The man who has never made a mistake has never made anything' GK Chesterton. Can we have done with 'spin' except from Graeme Swann on the cricket pitch!

For our administrators and regulators: back off. Have the confidence to allow people to develop their own ideas and run their own show. Be less risk averse and focus more on articulating risk more clearly (patients and public can and will understand this). Make targets clear,

patient relevant and important; resist the urge to tell us how to do it and focus instead on what should be achieved. Do all you can to encourage innovation and invention.

For innovators and inventors: send your best work to Thorax!

Competing interests AD and IDP are Joint Editors in Chief of *Thorax*, and are thus committed to making the Journal as excellent as possible. No other conflicts of interest.

Provenance and peer review Not commissioned; not externally peer reviewed.

Thorax 2011;66:91–92. doi:10.1136/thx.2010.157925

Who bears the costs of occupational asthma?

P Sherwood Burge

Most workers with occupational asthma who remain exposed to the causative agent have accelerated loss of FEV₁¹ and recover less when eventually removed from exposure than those removed within the first year of occupational asthma symptoms.² The reasons for this are not obvious, as most childhood onset asthmatics remain exposed to the causative allergens and infrequently develop severe airflow obstruction later in life. It is possible that the timing of exposure related to the maturity of the immune system is critical, and the development of a degree of tolerance is more common in childhood than later in life. Whatever the reasons, the recommendation for removal from exposure within a year of first occupational asthma symptoms is based on good evidence.²

There is an established societal view that those disabled by their work through no fault of theirs are entitled to some form of compensation. How this is achieved varies widely between countries. Most countries rely on an insurance-based system, sometimes with support from the government.³ In the UK there is a dual system with a no-fault compensation system without employer contribution funded by the

central government and a common law system which requires the establishment of negligence on the part of the employer. Neither work well in the context of occupational asthma. Central to compensation is the assessment of disability. Most disability systems work better when the disability is fixed (such as loss of a limb or irreversible airflow obstruction) than when the disability is very variable, as in asthma. Many occupational asthmatics are not really disabled in daily life, but are completely disabled from doing the job which caused their disease. There is therefore a strong case for directing compensation to re-entry into the job market. For a young person early in their career retraining with different exposures is often the best option, and some compensation schemes, such as in Finland and Quebec, facilitate this. Surveillance schemes mostly based on specialist clinic reports show the occupational asthma peaks later in life.⁴ Retraining in a different trade/profession then places the worker at the bottom of another career pathway with loss of income and promotion prospects. Many such workers would be better moved sideways/upwards where their exposures can be removed and their skills and knowledge retained. This should be more easily managed in large organisations (such as healthcare) but difficult for small employers, such as bakeries where occupational asthma developing later in life is fairly common.

There are therefore costs generated by the development of occupational asthma.

The employer loses a worker and perhaps production, has costs involved in replacement and retraining, and may have an increase in insurance contributions. The worker loses income, particularly in the third of workers in whom relocation fails and unemployment results.² Finding a new job often results in a lower income. Finally, the state usually provides financial assistance for those without work and income. Costs may be direct (such as healthcare, retraining and drugs) and indirect (such as loss of income and loss of productivity). These costs have been modelled in a paper by Ayres *et al* for typical UK male and female workers with occupational asthma due to isocyanates, flour or grain and latex or glutaraldehyde (see page 128).⁵ The methodology used the number of new notifications of occupational asthma to the SWORD surveillance scheme in 2003,⁶ a voluntary reporting scheme for respiratory physicians incorporating data from the occupational physicians reporting scheme OPRA, and estimated costs incurred over the lifetime of the disease from the point of first diagnosis (an incidence-based approach). The total lifetime costs for Great Britain in 2003 were then distributed between those incurred by the individual, the employers and the government. The costs were based on estimates from the literature rather than directly measured data, and included data from several European countries including the UK as well as USA and Canada. The average worker with occupational asthma was estimated to take about 4 days extra sick leave per year, with a quarter staying in the same job, a quarter being relocated with the same employer, 15% finding a job with a new employer and 35% remaining unemployed or retiring. Total lifetime costs were estimated

Correspondence to P Sherwood Burge, Occupational Lung Disease Unit, Birmingham Heartlands Hospital, Birmingham B95SS, UK; sherwood.burge@doctors.org.uk

between £94 000 and £198 000, being more for men than women, and more for workers sensitised to latex or glutaraldehyde than flour or grain. These estimates were extrapolated to the estimated 631 new workers with occupational asthma in 2003, giving lifetime costs for the 2003 cohort of £71.7 to £100.1 million. These figures are clearly estimates and may be some way from the true costs; however, the most interesting conclusion was the distribution of the costs between worker, employer and government, with the employer bearing only 3–4% of the total cost and the remainder being borne fairly equally between worker and government. Occupational asthma is clearly a bad disease for the worker and the government, the employer having little financial incentive to control the cause.

There are two main approaches to reduce the impact and costs of occupational asthma, either reducing the incidence or limiting its consequences. Occupational asthma is a preventable disease. Glutaraldehyde asthma in the UK has vanished at little extra cost, initially following limitation of glutaraldehyde use and then its replacement for cold sterilisation. The replacement of latex with nitrile and other materials for gloves was delayed while the costs of replacements decreased, latex asthma now being uncommon in UK medical practice.⁷ Both

these examples have taken many years from the identification of the problem to its control. Flour in bakers and isocyanates in moulders and painters have been more difficult to control; both still remain common causes of occupational asthma.

The medical consequences of occupational asthma are reduced by early removal from exposure, which can be enhanced by medical surveillance detecting early disease.² In the UK, occupational health is not part of the National Health Service, is not compulsory and when provided is managed by individual contracts between employer and provider. Many of these do not include management of surveillance failures⁸ contributing to the delay in diagnosis even when surveillance is in place.⁹ Reducing the impact of occupational asthma, and therefore reducing the lifetime costs, requires the return of the worker to employment without loss of productivity or income for the worker. For those who wish to work again, proper assessment of the workers' abilities and preservation of income while retraining is surely the way forward. This requires a change in many compensations schemes away from providing a regular pension for many years to compensation focused on support during retraining and return to work.

Competing interests None.

Provenance and peer review Commissioned; not externally peer reviewed.

Published Online First 20 November 2010

Thorax 2011;**66**:92–93. doi:10.1136/thx.2010.151506

REFERENCES

1. **Anees W**, Moore VC, Burge PS. FEV₁ decline in occupational asthma. *Thorax* 2006;**61**: 751–5.
2. **Nicholson PJ**, Cullinan P, Burge PS, *et al*. *Occupational asthma: prevention, identification and management. Systematic review and recommendations*. London: British Occupational Health Foundation, 2010.
3. **Dewitte JD**, ChanYeung M, Malo JL. Medicolegal and compensation aspects of occupational asthma. *Eur Respir J* 1994;**7**:969–80.
4. **Gannon PFG**, Burge PS. The SHIELD scheme in the West Midlands Region, United Kingdom. *Br J Ind Med* 1993;**50**:791–6.
5. **Ayres JG**, Boyd R, Hurley JF. The costs of occupational asthma in the UK. *Thorax* 2011;**66**:128–33.
6. **Ross DJ**. Ten years of the SWORD project. Surveillance of work-related and occupational respiratory disease. *Clin Exp Allergy* 1999;**29**: 750–3.
7. **Bakerly ND**, Moore VC, Vellore AD, *et al*. Fifteen-year trends in occupational asthma: data from the Shield surveillance scheme. *Occup Med (London)* 2008;**58**:169–74.
8. **Mackie J**. Effective health surveillance for occupational asthma in motor vehicle repair. *Occup Med (London)* 2008;**58**:551–5.
9. **Fishwick D**, Bradshaw L, Davies J, *et al*. Are we failing workers with symptoms suggestive of occupational asthma? *Primary Care Respir J* 2007;**16**: 304–10.

What's nice about the new NICE guideline?

John O'Reilly,¹ Michael Rudolf²

A new national guideline for the management of chronic obstructive pulmonary disease (COPD) was published by the National Institute for Health and Clinical Excellence (NICE) in June.¹ Although technically only applicable to England and Wales (and even then only if adopted by the Welsh Assembly), it will be perceived by many international authorities as 'the British guideline' (with apologies to our

Scottish colleagues), and it therefore seems appropriate to comment on what's new and relevant. It is over 6 years since the last NICE COPD guideline was published,² and it is important to note that this 2010 version is only a partial update, concentrating on various aspects of diagnosis and severity classification and the management of stable disease. The management of acute exacerbations was specifically excluded from the scope of the guideline revision. This has had the consequence of producing a lengthy document (the full web-based version is over 600 pages!), much of which will be regarded by many readers as out of date. The Guideline Development Group (GDG) and the publishers have gone to great lengths to make as obvious as

possible which parts of the guideline are new and which are not, but many will feel that this was a lost opportunity in not revising other sections as well.

One of the major strengths of a NICE guideline is that its recommendations are based on systematic reviews of the best available evidence (using extremely strict criteria for assessing the evidence), and also giving explicit consideration to cost effectiveness.³ In addition, the GDG is truly multidisciplinary, comprising healthcare professionals (doctors, nurses, physiotherapists) from primary and secondary care and patient representation.

It is good to see that the new NICE guideline now agrees with other international guidelines both in recommending the use of postbronchodilator spirometry for confirmation of diagnosis and also in using the Global Initiative for Chronic Obstructive Lung Disease (GOLD) classification of severity of airflow obstruction (table 1).^{4–5} It was always difficult to rationalise why, for example, a patient with forced expiratory volume in 1 s

¹Department of Thoracic Medicine, Aintree University Hospital, Liverpool, UK; ²Department of Respiratory Medicine, Ealing Hospital NHS Trust, London, UK

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