bicycle, or ski better without limitations of gas exchange!

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Correspondence to: Dr C M Doerschuk, Case Western Reserve University, Room RBC 787, 11100 Euclid Avenue, Cleveland, OH 44106, USA; cmd22@case.edu

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BOHRF guidelines for occupational asthma

A J Newman Taylor, P Cullinan, P S Burge, P Nicholson, C Boyle

Publication of the first evidence based guidelines for occupational asthma

New guidelines for the identification, management, and prevention of occupational asthma are published this month in *Occupational and Environmental Medicine*.[1] The first evidence based guidelines for occupational asthma, they were prepared by a working group that included clinicians, patients, occupational hygienists, and representatives of the Health and Safety Executive. The work was supported by a grant from the British Occupational Health Research Foundation (BOHRF). The guidelines will be supplemented by an abbreviated version for primary care practitioners, occupational health practitioners, employers, employees, and workplace safety representatives.

These guidelines are intended to increase awareness and improve the management of occupational asthma by all practitioners who encounter such patients, and to stimulate the means to reduce its incidence by those able to effect this.

The important issues in occupational asthma concern its aetiology, diagnosis, outcome and prevention. Questions about these are not readily answered by randomised controlled trials (RCTs) and, arguably, conventional hierarchies with the RCT at the apex are not appropriate for assessing the strength of evidence used in the generation of guidelines.[2] Although not having the high internal validity of the RCT, strong inferences can be drawn from observational studies (whose external validity can be greater than that of an RCT) when these are well designed and their findings consistent and plausible.

The guidelines address several questions that are of key importance to respiratory physicians:

- What proportion of asthma in adult life is attributable to occupation?
- What are the most frequent causes of occupational asthma and in which occupations are they encountered?
- What methods are most useful in the diagnosis of occupational asthma?
- How is a case of occupational asthma best managed?
- What is the prognosis of occupational asthma and what factors influence this?

WHAT IS OCCUPATIONAL ASTHMA?

Asthma can be aggravated or induced by an agent inhaled at work. ''Occupational asthma'' is generally limited to asthma induced by a workplace agent, either following the inhalation of an irritant chemical in toxic concentration (irritant induced occupational asthma) or, more commonly, as the outcome of a hypersensitivity reaction to an inhaled protein

Table 1 Agents responsible for more than 100 cases of occupational asthma (1992–7) and the occupations in which they were encountered[3]

Occupation	Agent
Spray painter	Isocyanates
Baker	Flour
Wood worker	Wood
Nurse	Glutaraldehyde
Laboratory technician, scientist and assistant	Laboratory animals
Welder, solderer or electrical assembly	Solder/colophony

allergen or chemical (hypersensitivity induced occupational asthma). Some 90% of cases of occupational asthma are hypersensitivity induced and the guidelines primarily address this group.

Irritant induced asthma typically develops within hours of an acute inhalation incident; hypersensitivity induced asthma only develops after a latent interval usually of several months—but occasionally weeks or years—of asymptomatic exposure. In some cases, usually when caused by inhaled proteins but also by some low molecular weight chemicals, hypersensitivity induced occupational asthma is accompanied by the development of specific IgE antibody which can be identified in the blood or by a skin prick test.

HOW FREQUENT IS OCCUPATIONAL ASTHMA AND IN WHOM DOES IT OCCUR?

Estimates of the incidence of occupational asthma by occupation and the agents identified as causative in the UK during the past 15 years have come from the Surveillance of Work and Occupational Respiratory Disease (SWORD) scheme to which chest physicians and occupational physicians have regularly reported new cases of occupational lung disease. The results of SWORD have been surprisingly consistent, with occupational asthma the single most frequently reported condition—accounting for some 25% of reported cases with an estimated average of 2000 new cases each year. These cases represent only those seen by chest and occupational physicians in whom the attribution of asthma to work is made and reported. Interestingly, with the exception of the rise and fall of asthma attributed to latex allergy during the 1990s, the other causes of occupational asthma and the occupations in which the cases were employed have remained surprisingly stable during this period. The agents identified as responsible for more than 100 cases between 1992 and 1997[3] and the occupations in which they were encountered are shown in table 1.

Several recent studies, many using data from the European Community Respiratory Health Survey, have estimated the proportion of cases of asthma in those of working age to which occupational factors have contributed. In essence, such studies estimate the relative frequency of different occupations in (usually) prevalent cases of asthma, comparing these with an occupational grouping considered not to have an increased risk from occupational cause. Two meta-analyses of these studies have recently been reported. The first suggested a median population attributable risk (PAR) of 9% for all studies and 15% for the highest quality studies; the second a PAR of 15%. The implication of these observations is that between 1 in 10 and 1 in 7 cases of asthma in adult life are, at least in part, attributable to occupation.

WHY MAKE THE DIAGNOSIS OF OCCUPATIONAL ASTHMA?

Many studies reported in the past 25 years have found that symptoms of asthma and airway hyperresponsiveness persist in a high proportion of cases (between a third and a half) followed up for several years after avoidance of exposure to the cause, which includes isocyanates, wood dusts, acid anhydrides, colophony, and snow crab protein. Although the majority of these studies followed up patients seen in hospital and are therefore likely to reflect more severe disease and those with continuing symptoms, they are consistent in indicating continuing disease in a significant proportion of cases. In addition, studies of social and financial consequences indicate that about one third of cases remain unemployed up to 5 years from diagnosis and suffer consequent financial disadvantage.

Several studies have identified common factors which increase the probability of a favourable prognosis. These include avoidance of further exposure to the causal agent; relatively normal lung function at the time of diagnosis; shorter duration of symptoms before diagnosis; and shorter duration of symptoms before avoidance of exposure. By implication, as emphasised in the guidelines, the resolution of symptoms and the restoration of normal lung function are most likely in patients whose occupational asthma is identified early in the course of the disease and who avoid exposure within a short interval from the onset of symptoms, ideally within a few months.

The high proportion of cases remaining unemployed after several years suggests that the value of a compensation scheme for occupational asthma could lie in supporting retraining, enabling return to the labour market. There is limited evidence from France and Canada to support this.

HOW BEST TO MAKE THE DIAGNOSIS OF OCCUPATIONAL ASTHMA?

The diagnosis of occupational asthma is an iterative process. The components that show the greatest diagnostic utility are: a history of work related symptoms; serial peak flow measurements; and, where applicable and available, identification of specific IgE antibody in the serum or by skin prick test. The gold standard for the diagnosis of occupational asthma is bronchial provocation testing which is indicated in a minority of cases and is of limited availability in the UK.

Sensitisation and the symptoms of occupational asthma due to the majority of causes of occupational asthma are most likely to develop in the first few years of exposure. Rhinitis accompanies IgE associated occupational asthma in a high proportion of cases and generally develops about a year before the onset of symptoms.

Because of the potential implications of occupational asthma for future employment, the guidelines recommend that the diagnosis should be based on objective criteria (functional, immunological, or both) and that suspected cases should be referred to a physician with expertise in the disease.

A history of symptoms of asthma which improve on days away from work, both by questionnaire and taken by experts, has a high sensitivity (few false negatives) but relatively low specificity (a high proportion of false positives). Objective tests are needed to increase the specificity of the diagnosis. Studies of serial peak flow measurements undertaken in a clinical setting have shown high sensitivity and specificity provided a minimum of four readings per day are made. Both visual analysis by experts and computed analysis show good diagnostic performance.

Pre and post shift measurements of lung function are of limited value and cannot be recommended in the diagnosis of occupational asthma. Increased airway responsiveness (using different methods) has been found in many cases of occupational asthma, but there are also many reports of normal airway responsiveness to inhaled histamine or methacholine within 24 hours of exposure in workers with confirmed occupational asthma. Airway responsiveness even within 24 hours of exposure is

not a sufficiently sensitive test and a negative result does not exclude a diagnosis of occupational asthma.

Specific IgE antibody is associated particularly with cases of occupational asthma caused by high molecular weight allergens such as enzymes, flour, latex, and laboratory animal urine proteins, but also with asthma caused by some low molecular weight chemical sensitisers including complex platinum salts, acid anhydrides, and some reactive dyes. Specific IgE confirms sensitisation to an agent encountered at work but, alone, does not confirm the diagnosis of occupational asthma. In general, specific IgE is very sensitive (few false negatives) but non-specific (high false positive rate). It therefore has more power in excluding an agent as a cause of occupational asthma than in identifying it as the cause.

HOW IS OCCUPATIONAL ASTHMA BEST MANAGED?

Because the risk of sensitisation and associated occupational asthma cannot be eliminated in those exposed to its causes, and the prognosis is best when the disease is identified and exposure avoided early, regular health surveillance is recommended for all workers where a residual risk of occupational asthma is identified.

Health surveillance should probably be undertaken at least annually and, because the risk of sensitisation and associated asthma is highest during the early years of exposure, more frequently in the first 2 years of exposure. Evidence for the effectiveness of health surveillance for occupational asthma is, however, limited to one study of isocyanate workers in Ontario, Canada where regular surveillance was combined with improved control of isocyanate exposure, making it difficult to dissociate their separate effects. Nonetheless, as would be anticipated, more cases of isocyanate asthma were identified initially; subsequently, in association with a reduced incidence, cases were identified sooner after the onset of symptoms with better lung function and a better outcome.

The implications for respiratory physicians are that they should endeavour to see and diagnose cases of occupational asthma within as short a time as practicable from the onset of symptoms and consider the potential for future employment with the patients' employers and their occupational health advisers.

HOW CAN OCCUPATIONAL ASTHMA BEST BE PREVENTED?

Although there is evidence for individual susceptibility—both genetic (atopy, HLA phenotype) and behavioural (smoking)—to several of the causes of occupational asthma, none is sufficiently discriminating to be appropriate as a basis for pre-employment screening. Studies of laboratory animal workers, bakers, enzyme detergent, platinum refinery and acid anhydride workers have documented gradients of increasing risk of sensitisation and asthma with increasing intensity of exposure, implying that the incidence of the disease would be reduced by a reduction in the level of exposure. The results of intervention studies have been reported: in a limited number of circumstances a reduction in the airborne concentrations of enzymes in the detergent industry, of latex in hospitals, and isocyanates in various workplaces have been followed by reduced incidence of disease.

CONCLUSION

It is hoped these guidelines will draw the attention of respiratory physicians and other health professionals to the importance of occupation as a cause of asthma in adult life, to the importance of accurate diagnosis at an early and potentially remediable stage of the disease, and to the need for communication between respiratory, primary care and occupational health practitioners and their patients' employers for optimal management of patients with occupational asthma.

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Authors' affiliations

A Newman Taylor, P Cullinan, Department of Occupational & Environmental Medicine, NHLI at Imperial College Faculty of Medicine, London SW3 6LR, UK
P S Burge, Birmingham Heartlands Hospital, Bordesley Green East, Birmingham B9 5SS, UK
P Nicholson, P&G, Whitehall Lane, Egham, Surrey TW20 9NW, UK
C Boyle, Industrial Chemicals Unit, Health and Safety Executive, Magdalen House, Stanley Precinct, Bootle, Merseyside L20 3QZ, UK

Correspondence to: Professor A J Newman Taylor, Department of Occupational & Environmental Medicine, NHLI at Imperial College Faculty of Medicine, Emmanuel Kaye Building, 1b Manresa Road, London SW3 6LR, UK; e.haining@rbh.nthames.nhs.uk

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