
Editorial

Preventing occupational asthma

What are the priorities for prevention of occupational asthma? Five years ago, it seemed difficult to prevent without further information on exposure-response relations obtained by prospective epidemiological studies incorporating measurement of environmental exposure.¹ This need is now accepted so that not only are such studies being carried out but it is also possible to view prevention with a broader perspective.

Prevention

Prevention is grouped into primary, secondary, and tertiary preventive activities. For occupational asthma, primary prevention means controlling the exposures that cause asthma. Secondary prevention is the detection of asthma at a sufficiently early stage that impairment and disability are minimised. Tertiary prevention is the provision of medical care of good quality to patients with asthma so as to avert complications.

Primary prevention

Prevention of the exposures that cause asthma starts with a clear concept of the two causal pathways to asthma at work. A sensitising agent may induce asthma; a high molecular weight agent may act alone or a low molecular weight hapten may conjugate with body proteins. Several hundred sensitising agents that can cause asthma are known.²

Secondly, heavy exposure to an inhaled irritant such as acetic acid may induce asthma.³ This was termed "reactive airways dysfunction syndrome (RADS)" when it was first described in 1985 by Brooks and colleagues.⁴ The term "occupational asthma" has implied sensitiser induced asthma to most British chest physicians and occupational physicians for many years. American usage is different and occupational asthma without qualification may mean not only irritant induced asthma but also asthma exacerbated by work. Harber⁵ has listed 12 asthma-occupation interactions and his article indicates some of the complications and uncertainties surrounding work, medical care, and asthma in the United States.

This editorial concerns itself primarily with sensitiser induced asthma. The control of sensitisers at work, and of allergic diseases, is a relatively new area.

The label "allergy" may lead to an erroneous assumption from industry that these diseases cannot be controlled by primary prevention because they are due to personal idiosyncrasy. The casuistically inclined could argue the same for lung cancer or pulmonary fibrosis.

To quote a recent text on preventing occupational disease and injury, "the central public health questions are what occupational exposures need to be controlled, when, and how. [Control involves] anticipation . . . surveillance, analysis, and control."⁶ The first step in prevention is, therefore, identifying what exposures to control. Because there are many sensitising agents, there are some similarities to controlling carcinogens.

Those in industry who are responsible for occupational health lack an accessible list of known sensitising agents because these are published in the specialist literature—for example, by Chan-Yeung in 1990.² An authoritative list, kept up to date, would be the obvious means for an employer to define work areas and processes with exposure to sensitisers and to define groups of exposed workers. This would provide a framework for other activities, such as product labelling, control technology, and medical assessments. The Health and Safety Executive has taken an initiative in drawing up a list that has been endorsed by the Health and Safety Commission. The "Indicative List" (a non-exhaustive list) has been published as part of a comprehensive package of measures, including an approved code of practice for the control of respiratory sensitisers, and was published for public consultation on 15 September 1992. Comments are invited for a three month period to arrive no later than 15 December 1992.

Methods of measuring high molecular weight allergens in air are still research procedures^{7,8} but concerns about methodology may divert attention from practical prevention. Corn⁹ has provided a general description of the assessment and control of environmental exposure with special reference to allergens. He lists 20 control methods. Fifteen relate directly to primary prevention: elimination, substitution, isolation, enclosure, ventilation, process change, product change, housekeeping, dust suppression, maintenance, sanitation, work practices, personal protective devices, waste disposal practices,

and administrative controls. He includes "medical controls" as a 16th, but many would not include the exclusion from exposure of workers deemed to be at special risk. Four are needed in order to carry out the remaining 16: education, labelling and warning systems, environmental monitoring, and management programmes.

Secondary prevention

If exposure to a sensitiser has been satisfactorily controlled there will be, by definition, no cases of occupational asthma. But controls can break down and employers need ways of detecting cases so that the environmental control strategy can be reviewed. Furthermore, there is evidence, reviewed by Chan-Yeung² that impairment and disability can be minimised by early diagnosis of occupational asthma and prompt removal from exposure.

There is no consensus on the best approach to detection of asthma in the workplace. Should symptoms be surveyed? If so, which questions should be asked? Would tests of pulmonary function such as measurement of bronchial responsiveness detect asthma at a presymptomatic stage? Should tests of immunological responsiveness to a sensitiser, such as skin prick tests, be carried out? Do they predict the development of asthma? Prospective epidemiological studies are needed to answer these important questions.

Secondary prevention practice in the United Kingdom varies from detailed screening procedures provided in the workplace to reliance on notification by the patient's general practitioner that a case has occurred. It may be not uncommon for procedures to be introduced without consideration of their likely effects or evaluation of their actual effects. Newill and her colleagues in the United States¹⁰ inferred that this was the case for pre-employment screening practices for animal allergy in the laboratory.

Whatever the secondary prevention procedure, it is important that the resultant action is timely and according to a plan familiar to the worker. If occupational asthma is confirmed, the appropriate action is to remove the patient from exposure by providing alternative work, to investigate his work area for lapses in existing controls, and to screen others with similar exposures as there may be other cases.

In the United Kingdom at the present time, investigation of the patient whose results in a screening test suggest possible occupational asthma is the province of the National Health Service and the Employment Medical Advisory Service supplemented where present by private occupational health services in industry. In this context of ready access to investigations, a case can be made for choosing a sensitive initial screening test because false positive test results should be identifiable at the stage of

definitive investigation in the occupational health clinic, general practice, or hospital. In countries where there is less access to medical care, a case could be made for specificity in screening tests although this will, naturally, mean that false negatives must be accepted.

Tertiary prevention

The National Health Service is responsible for medical care of the established asthmatic patient in the United Kingdom. Avoidance of further exposure is the major priority because exposure of a sensitised asthmatic person may result in death.¹¹ General guidelines for management change with professional opinion and recent ones have been published by the British Thoracic Society¹² and also by the National Institutes of Health in the United States.¹³

Surveillance

Surveillance programmes can describe how often occupational asthma occurs, its frequency relative to other occupational lung diseases, and the relative importance of different causes, and they can monitor trends in time. Since 1989, the United Kingdom has had information on asthma and other occupational lung diseases from the SWORD (Surveillance of Work Related and Occupational Respiratory Diseases) project.¹⁴ This national project, funded by the Health and Safety Executive, collects reports from chest physicians and occupational physicians. Of 2101 reports in 1989, the first year, the largest group was 554 reports of asthma. Isocyanates were the most common low molecular weight causal group (120 cases) and flour or grain the most common high molecular weight causal group (42 cases).

SWORD provides anonymous national information of value in setting priorities for research or for regulation by government or within companies. There are also other types of surveillance. The United States has SENSOR (Sentinel Event Notification System for Occupational Risks).^{15,16} Until now, this scheme has run in 10 states and six have included asthma as a target condition. SENSOR aims to link physicians with specialist resources for clinical and workplace investigation. It confirms the diagnosis of occupational asthma and intervenes to evaluate the workplace and to recommend controls. Patients and their workplaces are identified. The effects of intervention are immediate and local. It is likely that SWORD and SENSOR receive different data and have different effects because of their fundamental conceptual differences.

Pilot prevention projects

Evaluations of health care are still unusual in the United Kingdom although the recent changes in the NHS have stimulated debate.¹⁷ It is to be hoped that this will provoke interest in evaluative research in

prevention of occupational asthma in which new programmes are introduced on a pilot basis, with sufficient data collection so that their effectiveness can be monitored.

Irritants and asthma

The frequency with which irritant induced asthma occurs is unknown. The initial reports⁴ described asthma after high level, often accidental, exposures in which case it may be uncommon. On the other hand, it is possible that asthma exacerbated by irritants is responsible for the major burden of morbidity from occupationally related asthma. It is not clear if recurrent exacerbations of asthma increase the long term mortality or morbidity from asthma. These are important areas of uncertainty and worthy of further study.

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