Occupational asthma

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Occupational asthma is defined as variable airway narrowing which occurs as a result of exposure to an airborne sensitiser in the workplace. This definition does not include either worsening of pre-existing asthma due to work environment (although some authors believe it should) or airflow obstruction after exposure to an irritant – the so-called reactive airway dysfunction syndrome (RADS). Occupational asthma probably accounts for 2–6% of all asthma cases in the UK, although many believe this to be an underestimate. Common industries associated with occupational asthma are listed in Table 1, and the incidence of occupational asthma in workers in some high risk occupations is shown in Fig 1. Reports of other occupations or substances implicated in causing occupational asthma appear frequently in the literature.

Diagnosis

Diagnosing occupational asthma has consequences for workers, employers and the workplace, with significant financial and legal implications. When confronted with patients with suspected occupational asthma, it is therefore important to tackle three questions:

- Does the patient have asthma?
- Is the asthma work-related?
- Is the asthma caused by a specific sensitiser at the workplace?

Does the patient have asthma?

History and examination

A careful respiratory history is crucial. Particular attention should be paid to jobs which the patient has undertaken since leaving school. Information on work environment, substances used at work and methods of respiratory protection are important parts of the history. Symptoms tend to start months or years after the first exposure to the sensitiser. Cough, chest tightness, wheeze and reduced exercise tolerance due to breathlessness are the main symptoms, while conjunctivitis and rhinitis are common. Symptoms tend to worsen during work days and improve or disappear at the end of the working day, during weekends and holidays. Symptoms become more severe with prolonged exposure and take a longer work-free period to improve. Physical examination may reveal wheeze and coarse crackles, but often shows no abnormalities.

Investigations

- Spirometry is often normal, but it may show an obstructive defect with improvement after bronchodilators. An improvement of 15% in FEV₁ after bronchodilators suggests a significant asthmatic component.

Table 1. Substances and industries implicated in occupational asthma.

| Substance                      | Industry                                      |
|--------------------------------|-----------------------------------------------|
| Isocyanates                    | Plastic foam, paints, adhesives, marking ink  |
| Platinum salts                 | Platinum-refining industries, laboratories    |
| Acid anhydride, epoxy resin    | Adhesives, plastics, moulding resins, surface coating |
| Fumes from soldering flux      | Electronic industry                          |
| Proteolytic enzymes            | Washing powder, brewing, silk and leather     |
| Animals, including insects     | Plant and animal research laboratories        |
| Grain dust and wood dust       | Baking, milling, farm workers, carpenters     |
| Drugs (antibiotics, cimetidine)| Laboratory work in pharmaceutical companies  |
| Glutaraldehyde                 | Operating and endoscopy theatres, laboratories, cooling towers, leather tanning |
| Soya bean, tea, green coffee,  | Food processing                               |
| crustaceans                    | Welding, soldering                           |
| Fumes from stainless steel     | Persulphate salts and henna                  |
| Hairdressing salons            |                                               |

Fig 1. The incidence of occupational asthma in some high-risk occupations. (Adapted from Ref 3.)
• **Serial peak flow measurement** is the most important diagnostic procedure in occupational asthma (see below).

• **Bronchial provocation challenge.** If asthma is suspected, but spirometry and serial peak flow are non-contributory, some physicians perform bronchial provocation challenge with increasing concentrations of histamine or methacholine. This test identifies the minimal concentration causing 20% reduction of FEV₁ from baseline (Fig 2). A concentration of ≤4 mg/ml of histamine or methacholine is generally accepted as an indicator of hyperresponsiveness and aids the diagnosis.

**Is the asthma work-related?**

*Identification of relationship between asthma and work*

The history may be accepted on its own as diagnostic of occupational asthma if symptoms:

- started or worsened after the patient took up the job
- are worse at work and better during weekends and holidays
- are getting progressively worse in duration and severity when at work, and their resolution when patient is off work is becoming slower.

Serial measurement of peak flow over two weeks at work and two weeks off work is the single most important investigation for diagnosis of occupational asthma (Fig 3). It is preferable, although often not practical, to do the measurement at two-hourly intervals. The best of three blows is recorded. The relationship with times at work and off work needs to be clearly identified on the peak flow chart. If falsifying readings for the purpose of claiming compensation is suspected, a portable 'coded' peak flow meter may need to be provided. Decoding can be done later for all measurements by a computer package provided with the device. This method has the added advantage of showing the date and time of measurement, thus minimising errors in record keeping.

**Is the asthma caused by a specific sensitiser at the workplace?**

*Atopy*

A positive skin test to common allergens identifies non-specific atopy. A positive skin-prick test to the suspected substance and raised specific immunoglobulin (Ig) E are strong evidence of causation. It must be taken into account that IgE levels decrease, and may become undetectable 6–12 months after exposure has ceased.

**Specific inhalation tests**

The ‘gold standard’ for identifying the substance causing asthma is considered to be specific inhalation tests, but these are needed in only a minority of cases. They are indicated when:

- asthma is suspected, but the agent to which the patient is exposed is not previously recognised
- there is more than one recognised sensitiser in the workplace
- the diagnosis is still in doubt after other investigations have been carried out
- there are severe asthma symptoms and the need to establish that it is safe for the patient to go back to work
- the patient’s livelihood is at stake as a result of a diagnosis of occupational asthma.

Specific provocation tests should not be done for medicolegal purposes.
Specific inhalation tests should be performed in hospital under close supervision. Following inhalation, patients could be kept in a low dependency unit to record their peak flow, FEV₁, and forced vital capacity, and where medical help can be obtained if late-onset symptoms occur. The procedure is time- and space-consuming and should be done by trained staff. In patients with occupational asthma, the immediate fall in peak flow may be followed by a delayed and more prolonged drop (Fig 4).

Management of occupational asthma

The only effective treatment of occupational asthma is to avoid any further exposure to the sensitising substance. This may have significant implications for both employer and employee. The management plans should be discussed frankly with the patient who will ultimately make the decision. The medical management of occupational asthma is similar to that of non-occupational asthma.

Physicians are often not in a position to visit the workplace. None the less, it is their moral responsibility to raise concerns about work environment, especially if asthma is diagnosed in more than one person working in the same place. If the firm has or is attached to a medical service, the physician should approach it with the prior consent of the patient. Otherwise, physicians should seek the consent of the patient to pass their details to a professional body. The Employment Medical Advisory Service (EMAS) sets standards in the workplace, with particular emphasis on prevention. EMAS doctors are experienced in dealing with managers and trades unions and in inspecting places of work. They also inform employers of the legal implications of failing to apply preventive measures according to set standards.

Compensation for occupational asthma

Patients with occupational asthma are entitled to compensation through the DSS. The amount of compensation offered depends on the degree of disability caused by asthma and any loss of income incurred by having to change career.

Reactive airway dysfunction syndrome (RADS)

Brooks et al described RADS in 1981 and characterised its pathological features on bronchial biopsy specimens four years later. It follows exposure to an airborne irritant. Unlike asthma, symptoms start within hours of exposure and usually resolve within weeks, although they may last longer in some patients. Increased airway hyper-responsiveness is common, but this may be due to direct damage to respiratory epithelium, including nerve endings, rather than being true hyper-sensitisation. Features of RADS and asthma are compared in Table 2. Treatment of symptoms in RADS is similar to the treatment of asthma, with inhaled beta-2 agonists, anticholinergic agents and inhaled steroids being the mainstay of management.

References

1. Newman-Taylor AJ. Occupational asthma. Thorax 1980;35:241–5.
2. Meredith S, Norman H. Occupational asthma: measures of frequency from four countries. Thorax 1996;51:435-40.

Table 2. Comparison of clinical features of reactive airway dysfunction syndrome (RADS) and occupational asthma.

| RADS | Occupational asthma |
|------|-------------------|
| Caused by acute exposure to an airborne irritant | Caused by repeated exposure to a sensitiser in the workplace |
| Onset of symptoms within hours of exposure to the causative agent | Onset of symptoms within months or years from first exposure |
| Bronchial biopsies show desquamation and infiltration of mucosal wall with plasma cells | Bronchial biopsies show sloughing of epithelial cells, hypertrophy of bronchial muscles, mucosal infiltration with eosinophils and lymphocytes, and basal membrane thickening |
**Key Points**

- A careful history is key to the diagnosis of occupational asthma
- Serial peak flow measurement at work and off work is the most important investigation
- Specific bronchial provocation tests are not necessary for legal purposes
- The mainstay of management is to avoid further exposure to allergens

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8. Please fill in your full name and address on the back of the answer sheet in the space provided; this will be used to mail the form back to you after marking.

**Q1** The rising prevalence of allergic asthma is:

- a) associated with a reduction in specific childhood infections leading to dysfunctional immune programming and the development of allergies and asthma
- b) not associated with exposure to environmental tobacco smoke in utero and in early life
- c) primarily due to recent increases in exposure to atmospheric pollutants
- d) not related to recent changes in genetic factors
- e) associated with changes in lifestyle leading to increasing exposure to indoor allergens including house dust mite allergen (*Dermatophagoides pteronissinus*) and animal dander

**Q2** Asthma-related mortality:

- a) has been associated with increased use and reliance upon potent bronchodilators such as fenoterol in New Zealand during the 1960s and 1980s
- b) is increased in asthmatics with a recent A&E or hospital admission

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*Further details on CME are available from the CME department at the Royal College of Physicians (address above or telephone 0171 935 1174 extension 306 or 309).*

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3 Meredith SK, Taylor VM, McDonald JC. Occupational respiratory disease in the United Kingdom 1989: a report by the British Thoracic Society and the Society of Occupational Medicine by the SWORD project group. *Br J Ind Med* 1991;48: 292–8.

4 Bright P, Burge PS. The diagnosis of occupational asthma from serial measurements of lung function at and away from work. *Thorax* 1996;51:857–63.

5 Brooks SM, Lockley J. Reactive airway dysfunction syndrome (RADS). A newly defined occupational lung disease. *Am Rev Respir Dis* 1981;133(Suppl):1–3.

6 Brooks SM, Weiss MA, Bernstein K. Reactive airway dysfunction syndrome (RADS): persistent asthma syndrome after high irritant exposure. *Chest* 1985;88:376–84.

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