CORRESPONDENCE

Occupational asthma due to porcine pancreatic amylase

Editor,—We read with interest the report of Aiken et al. of a hospital laboratory worker who developed occupational asthma to amylase. We think that the method of investigation reflects the practice of respiratory physicians. We suggest that the approach by occupational physicians would be different.

This was an opportunity to understand, through a multi-cause investigation, the circumstances that led to an avoidable case of occupational asthma. It is only by understanding where appropriate controls failed that changes can be implemented to prevent further cases. The published study leaves us with many important but unanswered questions. What appropriate actions taken in line with the control of substances hazardous to health (COSHH) regulations and other United Kingdom regulatory publications? Were there effective guidelines for the health surveillance of hospital staff working with commonly encountered sensitizers, such as described by Smedley and Coggan, and are they in place now? Was air sampling taken at the workplace, and what airborne concentrations of enzyme were present at the time of the suspected work activities? Was the employee potentially exposed to any other respiratory sensitizers, and if so, why was amylase chosen as the agent for the bronchial provocation test?

Our other concern is that there are investigations which ought to be performed before considering whether a bronchial provocation test should be undertaken. We would argue against the use of bronchial provocation testing in establishing that amylase, or indeed any enzyme, is the cause of asthma unless there is significant doubt, as recommended in regulatory guidance notes. Enzymes are known respiratory sensitizers, their allergenicity is independent of the class of enzyme and is a feature of their protein content. Many studies have implicated amylase as a cause of occupational asthma in bakers and in pharmaceutical workers.11

A suspected case of occupational asthma should be investigated by history, before and after work spirometry, serial peak flow studies, and immunological testing (skin prick test or serology). None of these tests seem to have been performed in the reported case. We support the position that specific bronchial provocation tests should be avoided when a subject, with a history of occupational asthma, has:• Objective confirmation of asthma and work related bronchoconstriction
• Exposure to a well known agent of occupational asthma
• Confirmation of sensitisation to this agent detected by immunological tests.20

These findings are usually sufficient to establish the diagnosis of occupational asthma.21

Authors’ reply—Blain et al fail to distinguish the routine investigation of occupational asthma arising in a recognised setting from the diagnostic steps that are necessary when a novel cause is suspected (or a recognised causal agent seems to be relevant in an environment previously considered to be harmless). To adopt and paraphrase one of their critical comments, it is only by obtaining unequivocally positive results in unique situations from “gold standard” methodology that fundamental changes can be justified to prevent further cases. The importance of a definitive diagnostic outcome extends beyond resolving uncertainty in the individual case because there are wider issues of public concern. Thus in the present case, the clear practical demonstration that the new method for administering amylase was indeed the cause of the technician’s symptoms led to an alternative and safe method of enzyme delivery, and to a substantial improvement of his asthma; and the publication of our report has enabled this experience to be disseminated widely to an appropriate audience. It is difficult to see how further cases could be prevented more effectively, and it was clear from the reviewers’ comments that the report would not have been published had it not been for the unequivocal outcome of the bronchial provocation tests.

Although history, lung function measurements, and immunological tests (when available) all have a role in diagnosis they are not fully reliable, either individually or collectively, and return to work studies with peak flow monitoring are unblinded, unsupervised, and poorly specific (positive results may ensue simply from increased levels of exertion or colder ambient air, quite apart from subconscious bias or wilful distortion). Furthermore, the relatively crude measure of ventilatory function cannot readily be checked for technical quality. These conventional avenues to diagnosis are nevertheless adequate for the routine investigation of occupational asthma when it arises in recognised settings, as Blain et al indicated.

Although the disciplines of occupational medicine and respiratory medicine are necessarily different in some respects, we think that most occupational and respiratory physicians follow very similar approaches to diagnosis and management. Differences in emphasis are usually complementary, and close collaboration will generally provide obvious benefit. As it happens, the technician of our report had consulted an occupational physician initially. He was advised to use inhaled steroid and bronchodilator medication regularly, but no other action was taken. The general practitioner was concerned as to whether this advice was appropriate and so referred him to a respiratory physician for a second opinion. The respiratory physician thought it likely that the new method for covering historical material with amylase had caused occupational asthma, and he considered it important for this to be confirmed (or excluded)—hence our involvement to resolve this specific issue.

T C AIKEN
D J HENDRICK

Department of Respiratory Medicine and Regional Unit for Occupational Lung Disease, Newcastle General Hospital, University of Newcastle upon Tyne, UK

E T PEEL

North Tyneside General Hospital, North Shields, Tyne and Wear, UK

Correspondence to: Dr D J Hendrick, Department of Respiratory Medicine and Regional Unit for Occupational Lung Disease, Newcastle General Hospital, Westgate Road, Newcastle upon Tyne NE4 0BE, UK

NOTICE

Society of Occupational Medicine

Annual Scientific Meeting. 6–10 July 1998. St Catherine’s College, Oxford.

The meeting will comprise postgraduate seminars, plenary sessions, and work place visits. The meeting is recognised for CME, and PGEA approval is being sought.

Further information can be obtained from: Dr Donald G Bruce, Trident Medical Services, Building FT.1, AWE plc, Aldermaston, Reading, Berks RG7 4PR. Tel: 00 44 118 982 5974; Fax: 00 44 118 981 5329; email donald@bylands.deon.com
Occupational asthma due to porcine pancreatic amylase.

P G Blain and E Loughney

*Occup Environ Med* 1998 55: 434
doi: 10.1136/oem.55.6.434

Updated information and services can be found at: [http://oem.bmj.com/content/55/6/434.citation](http://oem.bmj.com/content/55/6/434.citation)

These include:

**Email alerting service**
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

**Notes**

To request permissions go to: [http://group.bmj.com/group/rights-licensing/permissions](http://group.bmj.com/group/rights-licensing/permissions)

To order reprints go to: [http://journals.bmj.com/cgi/reprintform](http://journals.bmj.com/cgi/reprintform)

To subscribe to BMJ go to: [http://group.bmj.com/subscribe/](http://group.bmj.com/subscribe/)