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Occupational lung disease

Investigating outbreaks

A J Newman Taylor

Precise identification of the specific cause of the disease is not necessarily essential to initiate effective control measures

In 1854, John Snow wrote on the outbreak of cholera in London:¹

“the most terrible outbreak of cholera which ever occurred in this kingdom is probably that which took place in Broad Street, Golden Square and the adjoining streets, a few weeks ago. Within two hundred and fifty yards of the spot where Cambridge Street joins Broad Street, there were upwards of five hundred fatal attacks of cholera in ten days”.

An outbreak is usually thought of as a sudden localised increase in disease incidence. Classically, outbreaks are the outcome of infection but, increasingly, non-infectious agents are identified as their causes. These may be newly introduced without recognition of the associated risk or a well recognised risk to which the level of exposure is not appreciated. Several examples have been reported in recent years: an outbreak of obliterative bronchiolitis in the workforce of a microwave popcorn factory in USA probably

caused by diacetyl, a volatile agent for flavouring butter;² outbreaks of organising pneumonia in Spain and in North Africa in textile spray workers which, in both instances, followed the introduction of a textile dye whose chemical formulation had been changed;³ and outbreaks of lymphocytic bronchiolitis and peribronchiolitis in workers in nylon flock production in the USA.⁴ In each of these cases the outbreak followed the introduction of a new chemical or process into industry. In contrast, an outbreak of asthma in a detergent manufacturing plant in UK in the late 1990s caused by allergy to enzymes was a recurrence of a problem well recognised in the late 1960s.⁵ The risk of occupational asthma caused by the inhalation of powdered enzymes used in detergent manufacture had been considered a problem solved for 25 years by encapsulation of the powdered enzymes into granules, a formulation introduced in the 1970s and which had been invariably used in this factory. However, the process used in the factory to add the enzymes to detergent probably

led to the break up of the granules into powder, recreating the circumstances of exposure of the late 1960s.

Outbreaks can be considered as failures of control; they are important to recognise, to investigate and their cause identified as the basis for remedial action. Identification of the cause of an outbreak of disease in a workforce is informed by knowledge of the classical epidemiological descriptors of time, place and person. What was the time course of the outbreak—did it follow the introduction of a new material or change in process? Was the disease more frequent in one part of the factory or in those working in a particular process? How did those who developed the disease differ from those who did not?

In this issue of *Thorax* Robertson and colleagues describe their investigation of an outbreak of allergic alveolitis (hypersensitivity pneumonitis) and occupational asthma in a car manufacturing plant in the UK (see page 981).⁶ While allergic alveolitis and asthma have previously been described in these workforces in the USA,^{7–9} this outbreak is the first reported in the UK and was larger, particularly in the number of cases of asthma reported. In one similar outbreak of allergic alveolitis in a car manufacturing plant in the USA, six cases of allergic alveolitis all worked in a machining area of the factory where they used a synthetic metal working fluid (MWF) which had been introduced 6–8 months earlier. All six cases, but not eight of nine workforce controls, had precipitating antibodies in their serum to *Pseudomonas fluorescens* which had been cultured from used MWF.⁷

The strength of potentially relevant associations, and therefore their informativeness, is diminished by misclassification and a specific (ie, minimise false positives) is preferred to a sensitive (ie, minimise false negatives) case definition. Similarly, where possible, objective evidence is desirable of potentially relevant factors such as the date of onset of symptoms, job title, tasks and site of work and dates of introduction of new or changed processes. The case definitions used in the study reported by Robertson and colleagues are clearly defined but, inevitably, are more specific for alveolitis than for asthma. The authors conclude that the most probable cause of the outbreak was contamination (probably microbial) of aerosolised MWF widely used in the factory. Consistent with this conclusion, those who worked in machining and used MWF from the largest common sump had a 3.5-fold greater risk of having disease (27.3%) than those working in other parts of the factory (7.9%), although this relationship was weaker for allergic alveolitis than for occupational asthma. The authors were reliant on questionnaire responses (and therefore personal recollection) for the date of onset of breathlessness, the largest number reporting this to have been March 2003 which preceded by 1 month a steep rise in tramp oil in MWF in the large communal sump as shown in the factory records.

While, on the basis of their findings, contamination of MWF seems the most probable explanation, as in the majority

of US studies the authors were not able to identify a plausible specific responsible agent. Levels of exposure to airborne MWF in the factory were generally below the current Health and Safety Executive guidance value. However, a number of micro-organisms were identified in MWF taken from the largest common sump, in particular *Acinetobacter* spp and *Ochrabactrum anthrophi*. 59% of cases of EAA had precipitating antibodies to one of these species or to used sump oil compared with 10% of cases of occupational asthma and 5% of asymptomatic (control) workers. The absence of an identified specific cause limits the potential for specific control measures, but is sufficient to warrant a focus on minimising microbial contamination of MWFs where these are used.

It remains unclear why this outbreak occurred. What led to a change, presumably in the microbial flora in MWF, sufficient to cause the outbreak? If future outbreaks occur, it would be interesting to make a comparison—not just between cases and non-cases in the factory with disease—but also between factories with disease (“case” factories) and factories also using MWFs but without cases of disease (“control” factories). Such comparisons could illuminate an underlying cause.

History tells us that precise identification of the specific cause of the disease is not essential to initiate effective control measures. Snow’s recognition that water contaminated with sewage in the Thames was the cause of the outbreaks of cholera

he investigated led to the initiation of effective measures to prevent the transmission of the disease many years before the identification of a bacterium as its cause.

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Postoperative mortality in lung cancer

Risk and benefit: the eternal Yin and Yang of thoracic surgery

Alex G Little

Quality of life, as well as its length, is an essential consideration in developing and recommending therapeutic strategies

With the occasional exception, non-small cell lung cancer (NSCLC) is not curable without some form of surgical resection. The good news—ie, the benefit—is that when appropriate oncological standards are followed, cure can be obtained in a substantial majority of operated patients. However, this benefit is not gained

without exposing the patient to risk. This risk/benefit relationship is the yin and yang—the two opposing considerations—of thoracic surgery. The proximate risks associated with lung resection are morbidity and perioperative mortality. The challenge, both for a given individual and for large populations, is to maximise the likelihood of benefit while minimising

the risk. The paper by Strand *et al*¹ in this issue of *Thorax* (see page 991) addresses the issue of operative risk with regard to 30-day postoperative mortality. This paper makes a major contribution to this issue by clearly defining specific risk factors and even developing a helpful model to estimate the risk of postoperative death.

This Norwegian study reviewed a total of 4395 patients who underwent some form of lung resection for NSCLC between the years 1993 and 2005. Considerable data were able to be reviewed because of the Norwegian law that newly diagnosed cases of cancer have to be reported to the Cancer Registry of Norway. Mining this database for patients with NSCLC revealed the following conclusions. While the overall 30-day postoperative mortality rate was 4.4%, the mortality decreased during the study period, demonstrating a continually diminishing risk of death. This rate seems appropriate as it is nearly identical to the