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Editorial

Compensating occupational asthma

Early in 1982, occupational asthma will become a prescribed disease in Britain under the terms of the Social Security Act (1975).[1] This is important legislation because occupational asthma is becoming increasingly common as society uses more and more respirable reactive chemicals in its industries. At the same time pneumoconiosis is becoming less common, and it is likely that it will soon give way to asthma as the most frequently compensated occupational disease of the lungs.

Compared with pneumoconiosis, there are a number of difficult problems that may confound attempts to define and recognise occupational asthma. The most obvious include the absence of a definitive chest radiograph and the fact that asthma is a common disease in the population at large. Coincidence alone may therefore be responsible for its development or recurrence in an individual industrial worker. In their report to the Secretary of State for Social Services, the members of the Industrial Injuries Advisory Council defined occupational asthma as "asthma which develops after a variable period of symptomless exposure to a sensitising agent at work". The probability of such development being related causally to the sensitising agent in question depends, of course, on the sensitising potency of the agent and its respirable concentration in the working environment. The Council took a conservative stance, and for the purposes of the Act prescribed seven groups of industrial agents that expert opinion considered the most clearly established causes of occupational asthma. These comprised: platinum salts, isocyanates, epoxy resins, colophony fumes, proteolytic enzymes, laboratory animals and insects, and grain (or flour) dust—agents that in general are potent sensitisers or are encountered at high levels of exposure. The Council recognised that additional agents may need to be prescribed under the Act, and recommended the situation be kept under continual review.

It has become popular in recent years to regard clinical asthma as the result of an interaction between (non-specific) bronchial hyperreactivity and a triggering factor.[2–5] Triggering factors may be intrinsic or extrinsic, and extrinsic factors may be specific (the individual is sensitised or exhibits an idiosyncrasy to a particular provoking agent) or non-specific (the individual and other asthmatics with the same degree of bronchial reactivity show similar susceptibility to numerous irritant stimuli). Both specific and non-specific triggers may be found in the workplace, and asthmatic reactions occurring at work may be the direct result of either. When there is a specific trigger, such as exposure to the respirable dust of platinum salts, few would quarrel with the resulting attack being defined as occupational, or with it being compensated. Attacks provoked by sub-toxic exposures to sulphur dioxide[6] or smoke[7] would not, however, be compensable under the present Act, nor would attacks provoked by work related exercise in environments where ambient air is cold[8] or dry[9] but not necessarily polluted. Few would quarrel with this either. The crucial point is that in the latter circumstances the occupational environment was not responsible for the primary asthmatic state—the fundamental development of bronchial hyperreactivity.

Bronchial hyperreactivity, as shown by an undue obstructive reaction to inhaled histamine or methacholine, is not observed in more than 5% of random samples of the general population.[10 11] It is a uniform though not exclusive finding in active asthma.[12 13] In some groups of workers exposed to platinum salts and proteolytic enzymes, as many as 50% have developed asthma;[14 15] and with all the agents prescribed under the present Act, prevalence estimates of 20% or more have been suggested in some situations. There is no reason to believe an extraordinary selection bias could have attracted disproportionate numbers of workers with pre-existing bronchial hyperreactivity into these industries, and it must be concluded that the occupational environment itself (presumably the specific agent known to trigger attacks) was responsible for this hyperreactivity. Since occupational asthma frequently arises in subjects who have never previously experienced any suspicion of asthma, this conclusion is not difficult to accept. It is supported by the observation that bronchial hyperreactivity often re-

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gresses when workers affected by occupational asthma change their jobs.[16] It is interesting that its degree has been noted to increase after late asthmatic reactions to bronchial provocation tests with a number of industrial agents responsible for occupational asthma.[17 18] Such increases typically follow viral infections also,[19] and to a lesser extent exposure to oxidising air pollutants such as ozone[20] and the oxides of nitrogen.[21] The occupational environment may consequently be relevant to asthma at three distinct levels: it may be primarily responsible for bronchial hyperreactivity developing (or increasing); it may provide specific sensitising agents that trigger attacks; it may provide non-specific triggering factors. In fact, increases in bronchial reactivity after sub-toxic exposures to oxidising air pollutants are rarely of clinical importance in subjects without pre-existing asthma,[22] and these pollutants are unlikely to be solely responsible when asthma emerges for the first time in an individual industrial worker.

When an industrial agent is responsible for both the development of bronchial hyperreactivity and the specific triggering of asthmatic attacks, there should be no difficulty in satisfying the terms of the new compensation legislation—providing the agent belongs to one of the seven prescribed groups. There may, however, be some difficulty in recognising the fundamental role of the occupational environment in the first place, particularly if the response pattern does not include symptomatic immediate reactions. Late reactions may not become apparent for some hours after an industrial worker has left the workplace, and may persist (or recur) for several days without further exposure.[23–25] A weekend away from work may not therefore produce an obvious improvement. Furthermore, exacerbations may appear to be directly provoked in non-occupational settings, particularly by non-specific irritant stimuli. This is to be expected if marked degrees of bronchial reactivity have been induced; and the question arises whether, for example, an inability to participate competitively in athletic pursuits because of exercise-induced asthma should be taken into consideration when assessing the extent of compensation. If the affected worker is atopic, it is also possible that degrees of hypersensitivity to common allergens that are ordinarily of little clinical consequence, could become important when bronchial reactivity is sufficiently increased. In an early case report of isocyanate asthma, asthmatic reactions to house dust and barn dust were prominent during periods of convalescence from occupationally induced attacks but not at other times.[26]

It has been suggested that the degree of bronchial reactivity undergoes a natural circadian rhythm,[27] the bronchi being at their most reactive during the usual period of sleep. This may influence both the severity and timing of occupationally induced attacks of asthma. Immediate asthmatic reactions may be provoked at lower threshold levels of exposure towards the end of the usual waking hours. The corollary is that the same exposure may be expected to produce reactions of different severity at different times of the day. A parallel phenomenon is that late reactions begin after a shorter latent period if the inciting exposure is towards the end rather than the beginning of normal waking hours.[28] The magnitude of these effects probably depends on the amplitude of the circadian change, which may vary considerably from subject to subject. Individuals showing the pattern of recurrent nocturnal reactions after a single occupational exposure may prove to be those with the greatest amplitude, the exposure itself possibly augmenting the circadian change.

Certain viral infections may produce appreciable increases in bronchial reactivity.[29] Occupational asthma, like non-occupational asthma, is consequently likely to worsen in the aftermath of such infections. In one interesting study of suspected isocyanate asthma, specific bronchial challenge tests provoked positive responses in some workers only during convalescence from viral infections.[30] At other times, the interaction of isocyanate sensitivity with lesser degrees of bronchial reactivity was presumably inadequate to generate clinically recognisable disease. It might be that occupational asthma is often manifested in its earlier or milder stages by intermittent symptoms occurring only with viral illnesses. Lingering symptoms of "bronchitis" after colds or influenza could thus have special significance in workers exposed to sensitising agents.[31] These are often disregarded, however—especially when they occur in smokers. The intermittent wheeze and cough of asthma are not readily distinguished from those of acute bronchitis, and it may be useful to examine the blood and sputum for eosinophils in these circumstances.

A vital but little studied aspect of the interrelationships between bronchial reactivity, asthmatic triggers, and their dependence on both occupational and non-occupational factors, is the fate of occupational asthma once exposure ceases. The Industrial Injuries Advisory Council took the balanced view that although specific sensitivity usually persisted, symptomatic asthma usually regressed. The evidence that specific sensitivity may persist rests largely on the observation that re-exposure often results in a prompt recurrence of symptoms, even after a single exposure. This implies that bronchial hyperreactivity has persisted also. If this is commonly the case, it remains an open question whether asthma induced occupationally might persist or recur in a purely

non-occupational setting. Experience with western red cedar induced asthma has produced disquieting data in this respect. After a mean period of 3·5 years without further exposure, only 38 of 75 affected workers were free of symptoms without medication, and the majority of these did have persisting bronchial hyperreactivity.[32] The probability of persisting disease was directly related to the durations of exposure before and after the onset of symptoms. The fate of other varieties of occupational asthma may well be different, particularly if symptoms usually arise after shorter periods of exposure and if a change in working environment rapidly ensues. With toluene diisocyanate induced asthma, the latency period before symptoms first arise is usually a few months only, and all possible combinations have been observed in its fate during the initial years after exposure ceases—persisting asthma; persisting bronchial hyperreactivity and isocyanate sensitivity without persisting asthma; persisting bronchial hyperreactivity without isocyanate sensitivity or asthma; and regression of all three.

Continual review is obviously important to assess the full significance of non-occupational factors that may modulate occupational asthma. Bronchial hyperreactivity may prove to be a predisposing factor among the small proportion of newly employed workers in whom it already exists. If so, there could be a screening benefit from pre-employment challenge tests with histamine or methacholine in those industries at high risk. In individuals in whom bronchial hyperreactivity does not already exist, it will be important to determine whether its induction precedes the development of clinical asthma, or whether the two arise simultaneously. If the former proves to be the case, tests of bronchial reactivity at regular intervals might be even more valuable in identifying the emergence of disease at the earliest possible moment. The full importance of this will depend on collaborative data showing that the ultimate fate of occupational asthma may be unfavourable in certain industries, and that it is indeed related to duration of exposure. A greater role may also be found for bronchial challenge tests with specific sensitising agents. The evidence obtained from such tests is often convincing, and must have influenced the Advisory Council in its initial choice of prescribed asthma inducing agents. If asthma arising in a laboratory worker exposed to rodents or insects is assumed to be occupational in origin and therefore compensable, it will be difficult denying similar compensation to workers in whom occupational asthma has been confirmed by specific bronchial challenge tests with, for example, wood dusts,[33 34] antibiotics,[35 36] or certain amines.[18 37 38]

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