LETTER TO THE EDITOR

Industrial injuries benefits for coal miners with obstructive lung disease

Professor Seaton criticises the approach adopted by the Industrial Injuries Advisory Council (IIAC) in its most recent report on chronic obstructive pulmonary disease (COPD) in coal miners. Among other things he is concerned that its recommendation, now accepted by the Government, that benefit should be awarded where miners with COPD have worked underground for 20 years or longer, even if there is no evidence of pulmonary coniosis on a chest radiograph, will lead to increased payments “to compensate smokers”, and that it will remove an incentive to the coal industry to control dust exposures. He also expresses concern that pressure will now be brought on the IIAC to reduce the qualifying period of employment to less than 20 years. As chairmen of the Council and its Research Working Group, we wish to respond.

The remit of the IIAC is to advise the Secretary of State for Social Security on matters relating to the operation of the industrial injuries scheme, and on the occupational diseases covered by it. The Council is prescribed for the purposes of social security compensation. While the prevention of occupational illness is a concern for all who work in this field, it is not the prime responsibility of the Council. It would be wrong in principle for the IIAC to withhold a recommendation for justifiable compensation because it might compromise efforts towards disease prevention. However, in practice we do not think that the IIAC's rules are likely to cause the problems that concern Professor Seaton. Other incentives operate to encourage safe working practices, such as the Health and Safety at Work Act and the threat of litigation.

We should also make clear that the IIAC does not make its recommendations in response to external pressures, but on the basis of scientific evidence. The Council is always pleased to receive representations from people with queries or suggestions about the industrial injuries scheme, and in many of its enquiries invites evidence from interested parties and from professionals with relevant expertise. In the final reckoning, however, its reports are based on the members' assessment of the available evidence.

With regard to COPD in miners, there is strong evidence that the incidence and severity of airways obstruction are increased both by smoking and by inhalation of coal mine dust. Furthermore, when both hazards are present in combination, the effects on FEV1 appear to be approximately additive. A difficulty arises, however, because the legislation underpinning the industrial injuries scheme treats diseases as “all or none” phenomena and does not cater explicitly for disorders, such as COPD, which range across a continuum of severity from what is only just abnormal through to major disability and premature death. To resolve this problem the IIAC set out to define the degree of airflow obstruction likely to cause clinically important disablement (a loss in FEV1 of approximately one litre), and then classified the disease as present if it exceeded this threshold.

The next step was to establish the circumstances under which, in a miner with COPD defined in this way and given smoking habits, the disorder could be attributed to work with reasonable certainty. This is the requirement of the law and has been interpreted as meaning “on the balance of probabilities”. It is mathematically equivalent to asking whether, given his smoking habits, the miner's dust exposure has been sufficient to double his risk of developing COPD. Of course, many miners with COPD attributable to dust on this basis could also have avoided their disease by not smoking. However, the same argument would apply to lung cancer caused by chromates or by work in tin mines, where again benefit is paid irrespective of smoking habits. Epidemiological studies carried out by the Institute of Occupational Medicine (IOM) and others indicate that, in both smokers and non-smokers, the cumulative exposure required to double the risk of disabling COPD is approximately 100–200 g/m².

The problem then was to translate this level of cumulative exposure into an index that could be applied when assessing individual claims. Two approaches were possible, one based on duration of work underground and the other on radiological evidence of dust retention in the lung. Neither was ideal. Exposure underground vary from pit to pit, seam to seam, and year to year, and simply counting years of work underground would fail to account for this variation. Although the presence of category 1 pneumoconiotic changes on the chest radiograph is statistically related to cumulative dust exposure, it is far from being a perfect marker. For example, in one large analysis carried out by the IOM, 64% of 4800 men with exposures exceeding 180 g/m² had radiographs classified as category 0. In the first place, the Council recommended an exposure criterion based on both duration of working underground (20 years) and a requirement for radiological evidence of dust retention in the lung. In retrospect this was perhaps unduly strict, but it had the merit of ensuring that compensation was available to many of the most deserving claimants.

Subsequently, reassessment came to light as described by Professor Seaton. The survey by Lewis and colleagues, although it had its limitations, indicated that coal mine dust was associated with loss of FEV1 in the absence of pneumoconiosis, a conclusion with which Seaton agrees. Also, a national analysis of occupational mortality found that, whereas death rates from pneumoconiosis in miners varied markedly from county to county, those from COPD were remarkably more uniformly elevated. This suggested that the requirement for category 1 pneumoconiosis on the radiograph might be discriminating unfairly against miners in some parts of the country. Seaton postulates that the explanation for the mortality findings lies in diagnostic transfer of COPD deaths to pneumoconiosis in areas where the latter has historically been common and states that, since the 1970s, death from pneumoconiosis has been vanishingly rare. Given that all deaths from occupational disease are referred to a coroner and are investigated at necropsy, this would imply high rates of error by pathologists in the areas concerned, and we are aware of evidence to support this contention.

A further problem was that some claimants, who had been refused benefit because they did not have radiographic evidence of pneumoconiosis, had later been shown to have the disease at necropsy, an anomaly which their families not surprisingly regarded as unfair.

In the light of these considerations the IIAC took the view that the requirement for radiographic evidence of pneumoconiosis as evidence of dust exposure should be dropped. The requirement for 20 years of work underground continues and is not arbitrary. It is an estimate of the duration of employment that in many mines would have led to a cumulative exposure of 200 g/m². With a lowering of dust concentrations in mines, calculation of the duration of exposure sufficient to double the risk of developing COPD may need to be revised, but we do not think this will be necessary for several years at least.

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NOTICE

International Society for Aerosols in Medicine (ISAM)

The 12th International Congress of the International Society for Aerosols in Medicine (ISAM) will take place on 12–16 June 1999 in Vienna, Austria. For further information contact the Congress Secretariat at the Vienna Academy of Postgraduate Medical Education and Research, Alser Strasse 4, A-1090 Vienna, Austria. Telephone: +43 1 405 13 8322. Fax: +43 1 405 13 8323. e-mail: medacad@via.at
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