Coalmining, emphysema, and compensation

“A second important factor in these cases is chronic bronchitis, which is present in a large proportion and really causes the chief symptoms. A third is the occurrence of emphysema, which is almost invariably associated with long-standing cases of pneumoconiosis.”

Thus in 1895 Osler described, from his own clinical and pathological observations, an association which has remained controversial ever since. Does exposure to dust in coalmining (and other industries) disable people by the production of pulmonary emphysema?

The diseases of coalminers have provoked almost as much argument since coalmining became the mainstay of the first industrial revolution as have their social conditions, and in the minds of many the two arguments have become inextricably linked. Today in the United States it is almost impossible to comment on the scientific investigation of coalminers’ diseases without being suspected of being in the pockets of one or other side in the sociopolitical debate. And yet the scientific debate can only be resolved by independent and objective investigation, the results of which should lead to betterment of the conditions under which miners work.

Although coalworkers’ pneumoconiosis was well described in Scotland in the early 1880s and appeared as “spurious melanosis” in the medical textbook used by my greatgrandfather in the 1840s, its existence was disputed in the British Medical Journal in the 1880s and studies by Greenhow and later Haldane gave rise to the widely held view in the early 20th century that coalmining was a relatively harmless occupation. Nevertheless, observations in the 1930s by pathologists, most notably Cummins, and radiologists in South Wales established once again the reality of coalworkers’ pneumoconiosis. Cummins went so far as to say in 1936: “...I can state positively that, in the great majority of pneumoconiotic lungs examined by me at Cardiff, marked emphysema has been a conspicuous feature.” In this classic paper, he discussed his studies of the elastic tissue of the lung, wherein he showed that the normal connections of the elastic network in pneumoconiotic lung were lost, thus allowing disruption and distension of alveoli by the unhindered distensive forces of the chest wall. By this time, a further argument had surfaced—namely, that the diseases now clearly suffered by coalminers were caused by quartz and that pure coal itself was harmless. This argument was settled by Collis and Gilchrist and by Gough in their studies of Cardiff coaltrimmers, whose pneumoconiosis was derived from exposure to quartz free coal.10 11

By the time of the second world war the full horror of pneumoconiosis and respiratory disability in coalminers had become apparent, and the Medical Research Council set up its pioneering pneumoconiosis unit in Cardiff under Fletcher, and later Gilson. The most notable achievements of this unit are undoubtedly those of Gilson and Cochrane, two medical scientists whose recent deaths have ended a remarkable era of pneumoconiosis research. Their work on the methodology of questionnaires, of lung function testing and radiology, and their application of these methods to respiratory epidemiology set the standards to which all their successors aspire; the application of these methods has led to the understanding of the interrelations of pneumoconiosis, emphysema, and dust exposure that we now possess. One important methodological advance, however, remained to be made before these relations could be properly investigated.

After nationalisation in 1947 the National Coal Board addressed the problem of coalworkers’ pneumoconiosis. Dr John Rogan set up the Pneumoconiosis Field Research, a prospective study ultimately of some 50 000 coalminers from selected collieries (representing the range of mineralogical and mining conditions in the industry) throughout Britain. The objectives were clearly stated—to determine how much and what types of dust caused pneumoconiosis, and what concentrations of dust should be maintained to prevent men becoming disabled by the air they breathe. Fundamental to this research was the development of techniques for measuring the exposure of miners to dust. This was achieved by grouping men according to common tasks and making measurements of the dust exposure of these groups. A dust sampler that selectively collected dust of respirable size (the MRE 113A
gravimetric sampler) was developed by Hamilton and Walton during the course of the research and remains in use worldwide as the standard instrument for monitoring dust concentrations in coal mines. Estimates of earlier dust exposure were also made, based on current measured exposure and detailed individual occupational histories. Questionnaires about respiratory symptoms were administered, 5-yearly chest radiographs and measurements of forced expiratory volume in one second (FEV₁) were planned, and the prospective research, including measurement of dust, was begun.

The primary aims of the research have now been achieved by scientists at the Edinburgh Institute of Occupational Medicine; the relations between exposure to dust, simple pneumoconiosis, and progressive massive fibrosis for dusts of differing coal rank have been defined, national coal-mining dust standards have been set to take account of these risk estimates. The complex interrelations between exposure to dust, lung function, and pneumoconiosis have also been unravelled. Coalminers show a fall in FEV₁ related to exposure to dust, and this effect adds to that of cigarette smoking. This fall has been shown in both cross sectional and longitudinal analyses of data and there is evidence that, as with the effects of smoking, it may be of clinical significance. Also, the risk of a miner's death being certified as having been due to chronic bronchitis and emphysema is directly related to his cumulative exposure to respirable dust during life. In a search for a plausible explanation for this, a pathological study of the lungs of miners who had, in life, taken part in the research and whose exposure to dust was, therefore, known showed that a miner's risk of having more than one 30th of his lungs affected by centriacinar emphysema also related to his lifetime cumulative exposure to respirable dust, after allowing for age and smoking histories. The relation with dust exposure was demonstrable only in lungs that also showed a fibrotic reaction to the dust, and it was specific to centriacinar emphysema—no similar association was found for panacinar emphysema, although the occurrence of both types of the disease was closely related to smoking and to ageing. More recent work has confirmed and strengthened these findings, demonstrating the same relations in the lungs of 95 lifelong non-smoking miners. In both of these studies there was also a clear inverse relation between extent of centriacinar emphysema and FEV₁.

Thus the observations of Osler 100 years ago and Cummins 50 years ago have been amply borne out by a careful combination of epidemiology, pathology, and environmental measurement. The evidence is so strong, in terms of exposure response and consistency, that a causative relation between coal dust exposure and emphysema seems likely. But is it plausible? I believe that it is. Cummins' observations on the elastic tissue of lung lead one to a biological explanation. Recent experiments in rats have shown that the immediate response to inhalation of coal dust at concentrations comparable with those to which miners have until recently been exposed is a neutrophil and macrophage alveolitis. This is associated with impaired leukocyte chemotaxis and with raised activities of proteolytic enzymes in the alveolar fluid, events that may be seen to be the first step towards both the accumulation of cells and dust that is the coal macule and also the biochemical breakdown of surrounding elastic tissue and alveolar walls that is emphysema. In view of these observations, it is perhaps not surprising that the pathological studies mentioned above showed that the dust related risk of having centriacinar emphysema was influenced by the amount of quartz in the dust—the more quartz the smaller the risk of emphysema at a given level of total dust exposure. This leads to a more general point, and an explanation of that which has been observed frequently but rarely investigated elsewhere where fibrosis occurs, is also emphysema. Sarcoidosis, chronic allergic alveolitis, fibrotic tuberculosis, even sometimes cryptogenic fibrosis, may all show a greater or lesser degree of emphysema as well. And the lungs of smokers with emphysema commonly show some fibrosis. There seems to be a common pathway to these two diseases, and coalminers' pneumoconiosis sits halfway between those characterised mainly by fibrosis and those predominantly emphysematous.

This, finally, brings me to the subject of so called "compensation." Clearly, many coalminers smoke, and develop respiratory impairment due to emphysema. It is of course usually impossible to determine with certainty whether exposure to dust or smoking is the main cause of emphysema in any one person, although it may be possible to argue this on the balance of probabilities. If all miners with reduced FEV₁, and appropriate disability were compensated, industry would be deprived of the opportunity of preventing the impairment by dust control as most of the disablement these days would undoubtedly be attributable to smoking. My predecessor at the Institute of Occupational Medicine and I have both argued that it is necessary to have some measure of a man's exposure to dust in order to determine the likelihood of his impairment being due to work rather than smoking on balance of probabilities. Because exposure to dust will not have been measured directly, a surrogate is necessary. Here the pathological studies quoted above provide a solution. Although the dust emphysema relation is only demonstrable in the presence of pathological pneumoconiosis, then it is reasonable to provide compensation for miners with early radiological (category 1/1+) International Labour Office classification signs of pneumoconiosis...
iosis who also have reduced FEV, and corresponding disability. This scheme should include both rounded and irregular opacities in category 1, as both are related to exposure to dust, it would minimise the twin injustices of compensation for disability induced by smoking and no compensation for disability induced by heavy dust exposure, though there is of course no way of eliminating all injustice from any scheme which divides a continuum arbitrarily into a dichotomy.

This line of argument was not accepted by the Industrial Injuries Advisory Council who did not appreciate its strength or logic when they called for more evidence. That evidence, in the form of the most recent work from the Institute of Occupational Medicine, is now available and is being sent to them. It will be interesting to see how they react.

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7 Haldane JS. The effects of dust inhalation. Transactions of the Institute of Mining Engineers 1917;55:264–73.