For Debate

Disability and Coal Workers' Pneumoconiosis

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British Medical Journal, 1974, 2, 652-655

Summary

The Industrial Injuries Advisory Council has reaffirmed the view that simple pneumoconiosis does not produce disability or shorten life. This is often true but, without overlooking the importance of chronic bronchitis, such conclusions are wrong in many instances.

Chronic bronchitis is probably commoner in miners without pneumoconiosis than in those with it, and an uneven distribution of bronchitis may mask the effects of pneumoconiosis. Cough and sputum in a miner with pneumoconiosis are not always due to chronic bronchitis. Disability is usually judged on measurement of vital capacity and forced expiratory volume, factors which cannot be expected to be significantly altered by simple pneumoconiosis alone. Other tests may show abnormalities which lead to ventilation and perfusion inequalities and to an increase in the ventilatory cost of exercise. Focal emphysema, often a consequence of simple pneumoconiosis, develops slowly and its influence on disability is delayed.

Disagreements arise because epidemiologists expect all lungs with simple pneumoconiosis to react in the same way, and they want a quantitative relation between simple pneumoconiosis and emphysema before attributing one to the other.

There are major difficulties in assessing disability but there is little justification for the regular application of the rule that if the results of ventilatory tests are normal disability is not present and if they are abnormal this is due to something other than simple pneumoconiosis.

Introduction

Interest in coal workers' pneumoconiosis has been low in recent years though there are about 40,000 certified cases in England and Wales and about 600 new ones every year. The Medical Research Council's Pneumoconiosis Research Unit in South Wales, which did such excellent work, especially in the 1950's, seems to have switched its interest to asbestos, perhaps in the belief that there is little more to be said about coal workers' pneumoconiosis. Fortunately the National Coal Board (N.C.B.) Medical Service is producing a series of epidemiological reports.

An individual physician cannot make much contribution except try to analyse what he sees happening in a controversial field. This is an attempt to do so and the time is opportune for

two reasons. Firstly, miners are no longer content with disablement benefit under the Industrial Injuries Act and several hundreds have made claims for damages against the N.C.B. alleging negligence because they have developed the disease. The stimulus for this was a settlement out of court for £7,500 of a claim brought in 1970. Secondly, the Industrial Injuries Advisory Council (I.I.A.C.) published a report in 1973¹ on the working of the Act.

The Present Position

Coal workers' pneumoconiosis occurs in two forms, simple and complicated. In simple pneumoconiosis there are multiple lung nodules which are classified according to their size (p up to 1.5 mm; m 1.5-3 mm; n 3 to 10 mm in diameter). The profusion of the nodules determines the classification into category 1 (limited distribution), category 2 and category 3 (affecting the whole of both lungs).

Some of those with simple pneumoconiosis develop the complicated form—progressive massive fibrosis. Fibrous masses larger than 1 cm in diameter become superimposed on the dust nodules and these may grow to produce extensive destruction. The development of progressive massive fibrosis rises steeply with the category of simple pneumoconiosis; it is uncommon with category 1 but it appears in about 30% of those with category 3. The cause is poorly understood but tuberculosis is sometimes a factor.

A miner who believes that he has pneumoconiosis may apply for certification. If a preliminary x-ray picture supports him he is examined by two members of the pneumoconiosis medical panel. If they diagnose category 2 or more he is certified. Assessment of disability is made in steps of 10%, and the vital capacity (VC) and forced expiratory volume in one second (FEV₁) are measured. If chronic bronchitis or emphysema is also diagnosed and considered to aggravate the disability caused by pneumoconiosis the disabilities may be added and benefit paid at the higher rate. This is automatic when disability attributed to pneumoconiosis alone is 50% or more.

As the transition from one category to another is smooth and subject to observer variation a difference of opinion between the miner's own doctor and the pneumoconiosis panel may arise. The latter's decision is not open to appeal but the man may reapply later. Dissatisfaction can be minimized by explanation about difficulties in classification, but this does not prevent discontent in a disabled man with x-ray changes regarded as somewhat less than category 2 who sees a colleague with no symptoms but more x-ray abnormality get benefit.

When a certified man dies a Coroner's postmortem examination is done and the heart and lungs are later examined by the pneumoconiosis panel. If pneumoconiosis is considered to have contributed materially to death the dependent will get benefit. Differences of opinion between the panel and the Coroner's pathologist or doctors acting for the dependent are frequent. Appeals go to a local tribunal of three laymen and afterwards to the Commissioner.

Many men with pneumoconiosis have no symptoms while others with mild or absent x-ray changes are disabled. To decide when symptoms and disability are due to pneumoconiosis or some other condition is often difficult, particularly when only simple pneumoconiosis is present. The official view is summarized in the Ministry of Social Security booklet *Pneumoconiosis and Applied Occupational Chest Diseases*.² This states that simple pneumoconiosis "is not itself a cause of disability. . . . In general, the lung function tests are within normal limits and there is no measureable impairment of lung function as a result of the disease. The expectation of life in cases of simple pneumoconiosis is not reduced." This implies that if disability is present or if a man dies with a respiratory disease the cause must be something other than simple pneumoconiosis. These views are accepted by the I.I.A.C. but there is a need for their critical re-examination.

Does Simple Pneumoconiosis Produce Symptoms?

Symptoms are commoner in miners than in non-miners and they have been analysed by the N.C.B. Medical Service with some curious conclusions. Ashford *et al.*³ related symptoms to age, smoking, and the presence or absence of pneumoconiosis of category 1 or more. As progressive massive fibrosis is much less common than simple pneumoconiosis the results can be safely applied to the latter. All symptoms were commoner in those with pneumoconiosis and this applied in particular to breathlessness, wheeze, and weather affecting the chest, whereas smoking was particularly associated with cough and phlegm. They concluded that the harmful effect of one of the two factors (simple pneumoconiosis and smoking) was not attributable to the other. They raised the possibility that men who knew they had pneumoconiosis tended to record symptoms indicating disability. If this was so it should have applied particularly to breathlessness. Smoking causes mucus hypersecretion, however, so it was no surprise that it was strongly related to cough and phlegm.

In the next report⁴ dust exposure was related to the presence of cough and sputum, which was classified as "bronchitis." Again there was a correlation between "bronchitis" and pneumoconiosis in all groups. A correlation between "bronchitis" and dust exposure was found in the 35-44 age group and in non-smokers aged 25-34 but not in others. The authors gave this association between dust and symptoms in a few groups as their most notable finding, but to me it was the strong association of pneumoconiosis and symptoms in all groups, particularly as the report excluded a desire to exaggerate as an adequate explanation.

In the third report⁵ men were divided into those with and those without chronic bronchitis by use of a questionnaire. Increasing dust exposure produced a decline in FEV₁. Simple pneumoconiosis was also associated with a decline in FEV₁, but after multiple regression analysis this was attributed mainly to the greater age and dust exposure of those with simple pneumoconiosis. As expected, men with pneumoconiosis differed mainly from others in having a much higher dust exposure; indeed they would not have developed one without the other. It therefore seems an odd statistical manoeuvre to load the expected reading for the FEV₁ by including a factor (high dust exposure) which may have affected those without pneumoconiosis in quite a different way by producing "true" chronic bronchitis. Heavy dust exposure may harm some by producing pneumoconiosis and others by producing bronchitis.

But the most unusual feature of the report is that the presence of cough and sputum in men with pneumoconiosis is equated with chronic bronchitis, without qualification. Even the inverted commas of the second report have gone and nowhere is there a reference to the first report. We may be seeing the creation of a myth that a person with cough and phlegm together with parenchymal disease invariably has chronic bronchitis. Is this to apply to sarcoidosis, fibrosing alveolitis, and even tuber-

culosis, or is it a distinction to be reserved for pneumoconiosis? A skilled clinician may be able to decide that chronic bronchitis is present in such circumstances, but a simple respiratory questionnaire has hardly reached that level of discrimination.

Does Simple Pneumoconiosis Produce Disability?

The official view that simple pneumoconiosis does not produce disability has already been quoted. It is based on many investigations, some of them measuring ventilatory capacity only. Such tests show lower readings in miners than in non-miners living in the same community. But in miners there is little difference between those without pneumoconiosis (category 0) and those with category 1 or 2. A reduction with category 3 is small⁶ or absent.⁷

There is an alternative interpretation for these findings which can destroy the official conclusions. Chronic bronchitis alters the pattern of dust deposition so that more is trapped in the airways and less deposited in the alveoli.⁸ Chronic bronchitis may protect people from developing simple pneumoconiosis so that there will be an excess of bronchitis amongst those with category 0 and a deficit in those with higher grades of simple pneumoconiosis. Because bronchitis exerts a profound effect on the VC and FEV₁, this may well explain why miners without pneumoconiosis have considerably reduced levels compared with non-miners, whereas those with simple pneumoconiosis show little or no further reduction. An uneven distribution of chronic bronchitis may mask a moderate effect of simple pneumoconiosis on ventilatory capacity.⁹ Strong evidence for an uneven distribution of major airways disease comes from Morgan *et al.*,¹⁰ who found that half their non-smokers with category 0 had asthma.

But can simple pneumoconiosis be expected to produce much reduction in VC and FEV₁? They are particularly reduced by extensive parenchymal disease or by narrowing of large and moderate sized bronchi. In most cases the amount of lung tissue replaced by simple pneumoconiosis is small so it is not surprising that ventilatory tests and lung mechanics are little affected. The coal nodules lie along the respiratory bronchioles so that if they do produce obstruction they will only do so at that level. Narrowing in distal airways produces little air flow obstruction, and it has been estimated that half can be completely blocked while resistance is increased by about 10%.¹⁰ Abnormalities at that level are best shown by alterations in the closing volume and by showing a reduction in lung compliance with increasing respiratory rates. Morgan *et al.*¹¹ have shown the presence of the latter in simple pneumoconiosis.

Therefore, instead of saying that a man with simple pneumoconiosis has no disability because his VC and FEV₁ are well preserved it would be more appropriate to say that if the readings are very abnormal (in the absence of progressive massive fibrosis or extensive coarse nodular simple pneumoconiosis) this must be due to some other condition. The tests at present used by panels for estimating disability due to simple pneumoconiosis are inappropriate, though there are no easy alternatives.

In regard to other measurements, the transfer factor is little affected overall but in those with very small nodules (p) it is often considerably reduced¹² and exercise produces excessive ventilation.¹³ In those without airways obstruction (a normal FEV₁:VC ratio) the residual volume and the ventilatory cost of exercise rises with increasing categories of simple pneumoconiosis. Some others have been found to have such abnormalities as pulmonary hypertension, a high alveolar-arterial oxygen gradient, hypoxaemia, and an increase in dead space:tidal volume ratio on exercise.^{11 14} Though these readings are normal in many patients there are several exceptions with considerable abnormalities which cannot be attributed to chronic bronchitis and smoking. Little change is found in lung mechanics except for reduction in compliance with increased rates of breathing, referred to above.

Do these abnormalities lead to disability? It has been claimed

that they do not, unless they are associated with other respiratory disease.¹¹ Most tests are carried out at rest and with short-term exercise. But what the miner often complains about is shortness of breath and exhaustion during a working shift. It is difficult to translate relatively small abnormalities of function shown in a laboratory to their effect on breathlessness during underground work in unpleasant and cramped conditions. Doctors seem to be less reluctant to accept an organic explanation for dyspnoea in—for example, angina—even in the face of normal haemodynamics in the laboratory.

These aspects were not considered by the I.I.A.C. and it accepted the present official position. Studies of the ventilatory cost of prolonged exercise may be the best way of clarifying the position.

Simple Pneumoconiosis and Focal Emphysema

If simple pneumoconiosis produces disability in some miners how does it do so? Obstruction in respiratory bronchioles, undetectable by routine tests, has already been discussed. This and the involvement of arterioles by the macules may produce ventilation-perfusion inequalities and dyspnoea. The development of focal emphysema may be more important.¹⁶ This is accepted as an entity due to pneumoconiosis in *Pneumoconiosis and Allied Occupational Diseases*² but during appeals doctors from the panels have cast doubts on this, putting forward Reid's¹⁶ views. She states that the dust is not necessarily associated with pathological emphysema, and this can be agreed. She also states that though the association of dust with emphysema suggests a cause and effect this does not often hold. She believes that these men have the centriacinar emphysema of non-miners in which dust becomes deposited in a harmless way. It would be surprising if this did not happen in some cases.

Typical focal emphysema is shown in fig. 1, with neighbouring normal acini without dust macules. The emphysema seems to be the result of a selective failure of dust clearance or a selective transport of dust into some scini. Probably even more important is a patchy and sometimes almost confluent dust-impregnated fibrosis and emphysema, giving an appearance similar to interstitial fibrosis. Another part of the same lung is shown in fig. 2. Ryder *et al.*¹⁷ and Lyons *et al.*,¹⁸ with their enormous experience, felt justified in classifying what is shown as focal emphysema. They described the postmortem findings in

FIG. 1—Section of lung showing focal emphysema. Acini without dust nodules have no emphysema. ($\times 3$.)

FIG. 2—Section of lung shown in fig. 1 showing smaller irregular dust foci and associated emphysema. ($\times 3$.)

247 cases and found much more emphysema in miners than in non-miners.

They have been criticized¹⁹ on the grounds that their cases might not have been representative, but there seems to be little substance in this so far as the postmortem findings are concerned. For practical reasons they did not include enough non-certified miners and their analysis was also made more difficult because in several certified cases the last radiograph before death was read as category 0 (these cannot be equated with category 0 in general). It was therefore not surprising that they found no correlation between radiographic category and emphysema. Caplan²⁰ studying similar subjects found more emphysema in those with category 0.

But is it necessary to find an increasing amount of emphysema with increasing categories of simple pneumoconiosis before accepting a causal relation between them? In the first place an excess of chronic bronchitis and related emphysema in those with category 0 may distort the results, and it is difficult to explain Caplan's²⁰ findings in any other way. Secondly, clearly simple pneumoconiosis does not always produce emphysema. There can be a variable response even within the same lung (see figs. 1 and 2). It may also be true that once sufficient dust has accumulated in those destined as a result to develop emphysema its extent and progression is little influenced by further dust deposition. Furthermore, the dust in the lung in fig. 2 seems unlikely to be as easily related to radiographic categories as the better defined nodules shown in fig. 1.

Focal emphysema develops slowly¹⁶ and its effects on ventilatory capacity may appear only many years after the causal simple pneumoconiosis. Working miners with pneumoconiosis may therefore show little reduction in ventilation, but a later study of the same men might show more effect. This may explain why Lyons *et al.*,¹⁷ with ventilatory tests done within four years of death, found greater impairment than Cochrane *et al.*⁷ Again there is good evidence that the p-type opacities are most commonly associated with emphysema.^{13 18} For clarification we need a further postmortem study of non-miners and miners with and without pneumoconiosis, and this should include histological assessment of chronic bronchitis.

Death Benefit

Cochrane *et al.*²¹ analysed the Death Benefit Scheme, but they

only considered advanced progressive massive fibrosis and concluded that panels were generous. Little difficulty arises in these cases but dissatisfaction is fairly common with less extensive disease. It could be argued that there is no justification for death benefit in such cases because Cochrane²² has shown their mortality to be the same as that of miners without pneumoconiosis. It is however, higher than in non-miners in the same community even when accidents are taken into account. This again leaves the possibility of more bronchitis in men with category 0 and a deficit in the higher categories of simple pneumoconiosis, which masks the harmful effects of the pneumoconiosis.

Personal experience makes it impossible to avoid concluding that simple pneumoconiosis makes a major contribution towards the death of some of those with cardiorespiratory disease. Arguments based on individual cases are countered by quotation of the handbook on the absence of disability and a normal life expectancy. In at least one area the panel almost invariably includes chronic bronchitis as a contributory factor, basing this on naked eye examination of material often received in an unsatisfactory condition. Statements about the extent and type of emphysema are also unreliable. When pneumoconiosis of the type shown in figs. 1 and 2 or coarse nodular simple pneumoconiosis (see fig. 3) is present it is hard to believe that it has not contributed to death.

FIG. 3—Section of lung showing extensive fibrotic nodular simple pneumoconiosis.

Difficulties are accentuated by two factors. Firstly, lungs are often not fixed in inflation, and secondly, members of pneumoconiosis medical panels are unreasonably expected to become competent pathologists almost overnight. They are recruited from many backgrounds and may have had no recent

experience with the ill and dying, and they may never have handled a human lung. Shortly after appointment they adjudicate on the part played by pneumoconiosis in the death of a man with complex cardiorespiratory problems.

A decision on death benefit is made by the insurance officer on the advice of the panel. Appeals go to a lay local tribunal whose members often admit their difficulties in choosing between differing medical opinions. Fig. 3 shows extensive nodular pneumoconiosis in a lung which also contained an area of progressive massive fibrosis 5 cm in diameter. In another case with pneumoconiosis of comparable extent the panel gave fibrosing alveolitis as the cause of death, whereas subsequent examination showed no evidence of this. In both cases the local tribunal rejected the views of the coroner's pathologist and preferred the opinion of the panel that pneumoconiosis was not a material factor. In a third case the panel only abandoned its view that death was due to a subdural haematoma at the hearing. Even then while admitting that death was, after all, due to respiratory causes, it excluded pneumoconiosis as a factor. These three cases, which came to the notice of one doctor in less than six months, are a cause for concern. Death benefit would have been lost but for the vigilance of trade union officials who sought further advice.

Representations were evidently made to the I.I.A.C. about the Death Benefit Scheme but no change was made. Indeed the only significant proposal, so far as coal workers' pneumoconiosis is concerned, was that benefit should not be paid automatically on diagnosis but only if disability was present. Sadly there is no guidance as to how mild but genuine disability can be determined. In the case of simple pneumoconiosis we are left with a rule of thumb which is usually used by the panel: if the ventilatory tests are normal, there is no disability; if they are abnormal, this is due to something else. It seems very unlikely that this is always true.

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