The answer to the second question is increasingly likely to be 'yes' towards each end of the scale.

Loss of binocular visual acuity is a greater loss in a man with poor eyesight because visual acuity falls from a higher level using both eyes to a lower level using one, even though the visual acuity in each eye singly was lower. Assuming the worst, in that when visual acuity is lost it will be lost from the better eye, any person with 6/18 or worse visual acuity (*i.e.*, corrected vision) in either eye should be regarded as being in need of special consideration, which may indicate extra protection from further damage to his eyesight where a significant hazard exists. In those employees who do not need

spectacles for correction of vision, well-fitting, wide plastic spectacles would be adequate in a chemical works. These should be worn at all times and further protection should be made available should an employee be specially exposed to danger from liquid splashes or irritant vapours or dusts.

We express our sincere gratitude to the two consultants for their valuable comments.

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Apparent Onset of Coal-Workers' Pneumoconiosis after leaving the Mines

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The case history is reported of a former coal-miner who, on admission to hospital with acute bronchitis, was found to have a chest radiograph suggestive of pneumoconiosis. However, the film taken on leaving the mines some four years previously was unremarkable, suggesting that the disease had either started or progressed during his four years away from the mines. Among the alternative explanations considered is the possibility that the diagnosis was in error and that the appearance was due to some radiologically similar disease which had chanced to affect an ex-miner. Some of the technical pitfalls inseparable from reliance on radiology are also mentioned as the appearance of progression may have been illusory. Other possibilities are that the disease was not the simple pneumoconiosis of coal-workers but some other form of pneumoconiosis, such as Caplan's syndrome or silicosis, in both of which the natural course of the illness may be quite different. Nevertheless, although it is clear that coal-workers' pneumoconiosis does not normally progress once the subject is removed from the mines, the possibility remains that in a small number of cases it may do so.

One of the recurring problems which confronts clinicians is the patient whose chest radiograph is studded with miliary nodular opacities but who has few symptoms referable to this finding. Quite often examination and investigations prove unrevealing but a history of occupational exposure to a dust known to produce this radiographic appear-

ance is elicited, and this is welcomed as providing a diagnosis. Unfortunately, sometimes in this situation the patient further reveals that since leaving contact with the dust he has had radiographs which have apparently been normal, and the question then arises whether the radiological changes can in fact be attributed to pneumoconiosis.

The present paper describes such a case in a coal-miner who left the coal-face with a compar-

FIG. 1. Chest radiograph, 1961.

FIG. 2. Chest radiograph, 1965.

actively normal chest radiograph (Fig. 1) and yet developed the picture of pneumoconiosis a few years later (Fig. 2), and who presented the opportunity for considering the possible explanations of this unusual sequence of events. This, it is felt, is a clinical paradox quite frequently encountered and, as the contraction of the coal-mining industry forces more and more miners to seek other employment, it is likely to become more so. This paper emphasizes the fact that absence from the pit-face for a number of years does not entirely preclude the diagnosis of pneumoconiosis, and that such a diagnosis should not be rejected because the subject has previously been declared to have a normal chest radiograph. Some mechanisms whereby such a situation may arise are discussed, although in the individual case it is seldom possible to be certain which explanation is the correct one.

Case Report

In September 1965 a 30-year-old soldier was admitted to hospital with an acute episode of bronchitis and his chest radiograph (Fig. 2) showed numerous coarse miliary opacities scattered throughout both lung fields. He made a rapid recovery with simple symptomatic measures alone and, apart from a similar incident six months later, remained well until his subsequent discharge from the Army, although the radiograph remained unchanged. There were no physical abnormalities, the sedimentation rate was normal, and sputum cultures for acid-fast bacilli were negative. He had never complained of pains in the joints, neither was there evidence of joint disease: the differential agglutination titre was negative at a dilution of 1:5. In spite of the extensive radiological lesion he was not unduly dyspnoeic on exertion, and the ventilatory volumes and carbon monoxide transfer factor were normal: the vital capacity was 5.11 l. and the F.E.V._{1.0} 4.3 l., and the transfer factor (continuous breathing method) was 23 ml./min./mm. Hg at rest and 39 ml./min./mm. Hg on exercise. The occupational history was that he had worked at the coal-face in Durham for almost nine years and during this period had had six-monthly miniature mass radiography (M.M.R.) films for which he was never recalled for further examination. Just a few weeks before leaving the mines he had had a full-plate chest radiograph which, as far as he knew, had not aroused any adverse comment (Fig. 1). In 1961 he left the mines and a year or so later he applied to join the Army in which he had served continuously since.

Discussion

There is no laboratory investigation which will establish the diagnosis of pneumoconiosis; it is therefore to some extent arrived at by exclusion. Also, there is no specific treatment for pneumo-

coniosis, unlike certain radiologically similar conditions, for instance, tuberculosis. Other disorders which may be indistinguishable on the radiograph, for example carcinomatosis, require positive diagnosis on account of their grave prognostic significance. For these reasons one hesitates to attribute the mottled appearance of the lung fields in this type of patient to pneumoconiosis when it may be due to some other disease unrelated to the industrial history. In particular, in a well patient it is impossible effectively to exclude sarcoidosis. The fact that there is no hilar adenopathy and no defect of gas exchange does not rule it out: but if the radiograph suggests pneumoconiosis, and the clinical features fit in with pneumoconiosis, and the subject is a candidate for pneumoconiosis, the possibility that he has pneumoconiosis merits examination.

Even if the diagnosis is accepted, it is permissible to question whether the apparent changes in the radiograph in fact reflect a corresponding change in the disease. In those cases where in the past M.M.R. films only have been scrutinized, it is impossible to be sure that the shadowing has not been missed, as it is well known that this type of film does not demonstrate the reticular pattern at all well. Even when full-plate films are available, it was shown by Fletcher and Oldham (1949) and recently emphasized by Wise and Oldham (1963) that technical differences of processing can greatly change the appearance of early cases and thus account for diagnostic failure. This is especially pertinent in the present case, for not only were the two plates taken at different centres but they are unfortunately both of indifferent quality, the earlier one in particular being unsatisfactory for assessing pneumoconiosis. The subsequent development of focal emphysema can also give rise to a deceptive change in the radiograph as the nodules become more discrete and dense by contrast with the surrounding hyperinflated lung, and this may bring to light lesions which are so slight that they were not previously apparent. This is only reflected in the functional abnormality of an increase in the alveolar-arterial oxygen difference which becomes progressively more marked as nodulation increases in size and extent. All these sources of error make the task of those responsible for reading the films very much more difficult.

In the present case, the radiological evidence of progression appears to be moderately strong, and this raised the objection that it is widely taught that anthracosis uncomplicated by progressive massive fibrosis does not progress after cessation of exposure (Perry and Holmes Sellors, 1963; Hunter, 1962).

By implication, therefore, anyone leaving the coal-face with a clear radiograph will not develop the disease. There is, however, one situation in which the background of simple pneumoconiosis is slight or absent and the opacities often appear with a suddenness that is not usually observed; that is in rheumatoid pneumoconiosis or Caplan's syndrome (Caplan, 1953; Caplan, Payne, and Withey, 1962). Although, as originally described, the opacities were between 0.5 and 5 cm. in diameter, the concept was subsequently expanded to include the appearance, sometimes encountered, of multiple peripheral, discrete, well-defined nodules between 0.3 and 1 cm. in diameter. The same authors also recognized that the lung lesions could appear after the onset of arthritis, coincident with it, or as much as 10 years before it, but that in the latter case the sensitized sheep cell agglutination test was sometimes positive. Although in the present case this investigation was negative, the radiological features are suggestive of Caplan's syndrome. However, the size of these nodules, which is at the lower end of the acceptable spectrum, their silent appearance some years after the patient had left the mines, and the lack of clinical or serological manifestations of rheumatoid disease seem to require an expansion of the syndrome beyond the point where it is usefully recognizable in the absence of histological proof.

Confusion may also arise because the pathological changes in coal-miners may be predominantly those of silicosis rather than anthracosis, and the former is thought to progress despite the cessation of exposure and even to appear *de novo* some years later (Mavrogordato, 1926; Montesano, 1947). Unfortunately, the radiographs may not be helpful in distinguishing the type of dust inhaled, but silicosis is not a prevalent occupational hazard in the mines where the present subject had been employed.

Finally, it is by no means certain that uncomplicated coal-workers' pneumoconiosis does follow its traditional benign and non-progressive course once the environment is changed. The point is an important one, for it raises the question whether removal from dust exposure influences the disease

at all. Davies, Fletcher, Mann, and Stewart (1949) presented formidable evidence in favour of removing affected miners from their environment when they examined pairs of radiographs of affected subjects taken at intervals of from five to 14 years. Those who had continued underground for at least half the intervening period showed an increase in the number of opacities, whereas the 81 men who were removed as a result of the first film showed no increase in the number or size of the opacities (although progressive massive fibrosis developed in a few). Support for this view came from Cochrane, Fletcher, Gilson, and Hugh-Jones (1951), who examined radiologically 75% of the workers in a colliery after an interval of two and a half years and found that progression of simple pneumoconiosis only occurred in men who had continued to inhale the dust during the intervening period and never in those who had been away from mining during this time. However, there have been conflicting reports, and Stewart (1948) and Lieben, Pendergrass, and Brieger (1965) concluded that the condition frequently continued to progress after the cessation of exposure. If this is so, it seems possible that a radiologically negative or 'preclinical' case may develop the typical appearance during the ensuing years.

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