

Coal Workers' Pneumoconiosis

WILLIAM KEITH C. MORGAN

To cite this article: WILLIAM KEITH C. MORGAN (1971) Coal Workers' Pneumoconiosis, American Industrial Hygiene Association Journal, 32:1, 29-34

To link to this article: <http://dx.doi.org/10.1080/0002889718506403>



Published online: 04 Jun 2010.



Submit your article to this journal [↗](#)



Article views: 7



View related articles [↗](#)



Citing articles: 8 View citing articles [↗](#)

Coal Workers' Pneumoconiosis

WILLIAM KEITH C. MORGAN, M.D.

Appalachian Laboratory for Occupational Respiratory Diseases, Bureau of Occupational Safety and Health, U. S. Public Health Service and Department of Medicine, West Virginia University School of Medicine, Morgantown, West Virginia 26506

☞ The medical indications of a specific pneumoconiosis resulting from inhalation of coal dust are presented. The characteristics of simple pneumoconiosis and of progressive massive fibrosis are discussed. The effects of cigarette smoking obscure and complicate the respiratory change induced by coal workers pneumoconiosis. The etiology of the disease is certainly associated with the inhalation of coal dust, therefore prevention of the disease can be achieved by control of such dust exposures.

Introduction

IN CONTINENTAL EUROPE, the United Kingdom, the United States, Australia and South Africa, coal is still extensively mined and remains one of the cheaper sources of energy. In most of these countries it is accepted that coal miners have a higher prevalence of lung disease than does a comparable population of non-miners. The cause of this increased morbidity and mortality from lung disease, however, is not completely understood and, moreover, cannot be attributed to a single respiratory disease or to any one particular set of circumstances encountered in job performance. Thus, while most authorities accept the fact that there is an increased prevalence of non-specific obstructive airway disease in coal miners as a whole, there is at present no conclusive evidence to show that it is related to the inhalation of dust except in special circumstances.

Until very recently, coal workers' pneumoconiosis went by the name of silicosis in the United States; a state of affairs that inevitably led to much muddled thinking. Informed opinion here is at last recognizing that coal workers' pneumoconiosis and silicosis are distinct entities. Perhaps a brief review of the evidence to show that coal workers' pneumoconiosis is a disease in its own right is apropos at this stage.

While coal workers' pneumoconiosis is predominantly associated with underground coal mining, it was the occurrence of the disease in coal trimmers that first called attention to its existence as a separate entity. A coal trimmer is a special type of stevedore who is responsible for the loading and distribution of coal in the holds of ships. Unless the coal is evenly distributed, the ship may become unstable and develop a list while at sea. Exported coal is washed prior to being loaded and its silica content is minimal. Collis and Gilchrist¹ and later Gough² showed that Cardiff and Swansea coal trimmers developed radiological abnormalities which were either identical to or bore a close resemblance to classical silicosis. At post-mortem their lungs were found to be full of coal dust, however, the silica content was not increased above the general non-mining population. Moreover, it was later shown that the prevalence of coal workers' pneumoconiosis, and this includes both progressive massive fibrosis and the simple variety, bore no relationship to the silica content of the coal mined. On the other hand, the presence of radiological changes in life correlates well with the coal dust contents of the lungs found at postmortem. Additional support for the hypothesis that coal dust or carbon alone can produce disease was forthcoming from the demonstration of a type of

pneumoconiosis that developed in carbon electrode workers as a result of exposure to pure carbon.³ This was pathologically and roentgenographically similar to coal workers' pneumoconiosis. The silica content of the lungs of these persons was either less or the same as that found in the general population. There would thus seem to be little doubt that coal workers' pneumoconiosis is distinct from silicosis.

Pneumoconiosis is defined as a diagnosable condition produced by the inhalation of dust. Coal workers' pneumoconiosis is therefore caused by the inhalation of coal dust. It has been classified into two types, simple and complicated, and the diagnosis and differentiation depends on the radiological appearance of the chest film.

Simple Pneumoconiosis

Simple pneumoconiosis is recognized by the presence of either multiple small pinpoint and nodular densities or reticulation in the chest film. This variety has been further subdivided into categories 1, 2, and 3 according to the extent and distribution of the nodular opacities. Pathological studies have shown that these densities represent small aggregates of coal dust with some surrounding fibrosis.

These collections of coal dust are located around the first division of the respiratory bronchiole. They are sometimes accompanied by a little reticulin fibrosis and in category 2 and 3 simple pneumoconiosis there may be a mild to moderate dilatation of the respiratory bronchiole—so called focal emphysema. The latter is a consequence of atrophy of the smooth muscle in the bronchiolar wall. The gross appearances thus closely resemble centrilobular emphysema; however, unlike this entity the respiratory bronchioles do not show evidence of bronchiolitis.

Respiratory impairment is found only in category 2 and 3 simple pneumoconiosis and even then is seldom associated with disability. As indicated earlier, miners as a whole have more respiratory symptoms and a lower ventilatory capacity than do non-miners. At first glance this would seem to incriminate their occupation. On the other hand, when the

ventilatory capacity of a group of miners with radiologically evident coal workers' pneumoconiosis is compared to a comparable group of miners with no radiological evidence of the disease, several interesting anomalies are evident. First, there is no difference in ventilatory capacity between miners with category 1 and 2 coal workers' pneumoconiosis and those without it. However, those who have category 3 do show a slight but significant decline in their ventilatory capacity. When progressive massive fibrosis is present, there is a significant and marked decline in pulmonary function (Figure 1). It is thus apparent that simple pneumoconiosis produces little or no effect on ventilatory capacity until category 3 is reached. It might be argued, however, that the disease might affect other aspects of pulmonary function, and indeed there is some evidence to this effect. Both Hugh-Jones⁴ and Brasseur⁵ have demonstrated an increased A-a gradient in miners with simple pneumoconiosis; but in this connection both workers stress that this finding is not associated with either dyspnea or pulmonary disability. Arising out of this finding, Lyons and his co-workers were able to demonstrate a reduction in the diffusing capacity in subjects with the pin-head variety of simple pneumoconiosis but not in the nodular type.⁶ The recent claims that coal workers' pneumoconiosis leads to "small vessel" disease and

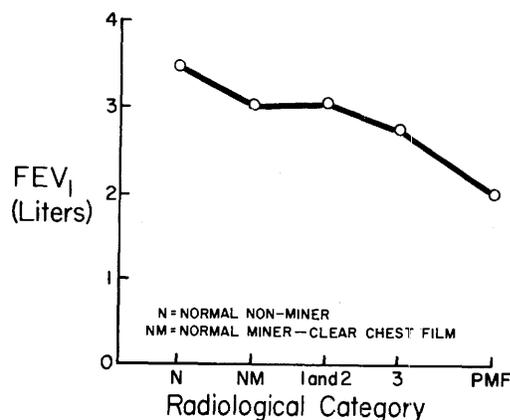


FIGURE 1. Relationship of radiological category to the forced expiratory volume in one second (FEV₁).

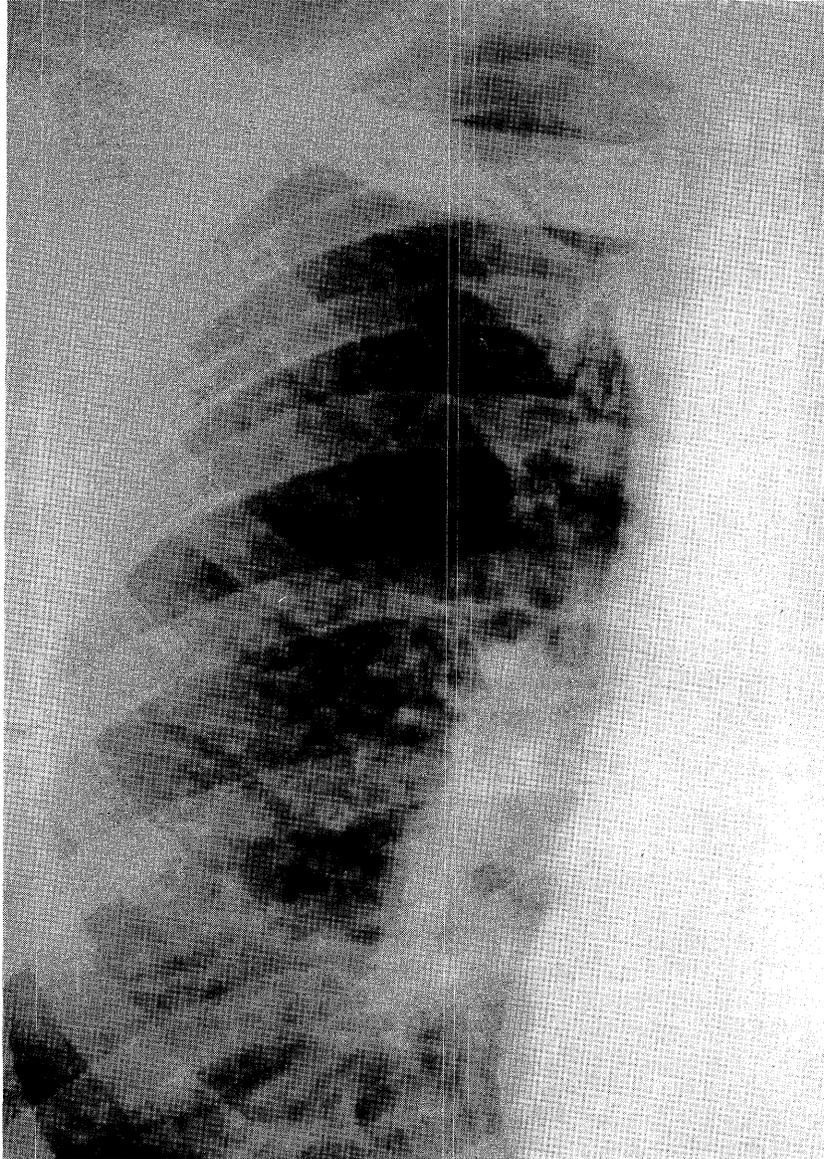


FIGURE 2. Close-up view of a radiograph of the right lung of a subject with simple pneumoconiosis.

pulmonary hypertension have not been confirmed. Furthermore, the suggestion that this type of physiological impairment may be found in the absence of radiological evidence of the disease seems highly improbable, since it is reasonable to assume that in general the more dust present in the lungs, the greater the likelihood of physiological impairment.

It has been repeatedly shown that the greater the amount of dust in the lungs, the higher the radiological category; thus it can be inferred that those with clear chest films have little in the way of dust retention and are most unlikely to show respiratory impairment. In this connection, it is pertinent to note that the British regard category 1 simple pneumo-

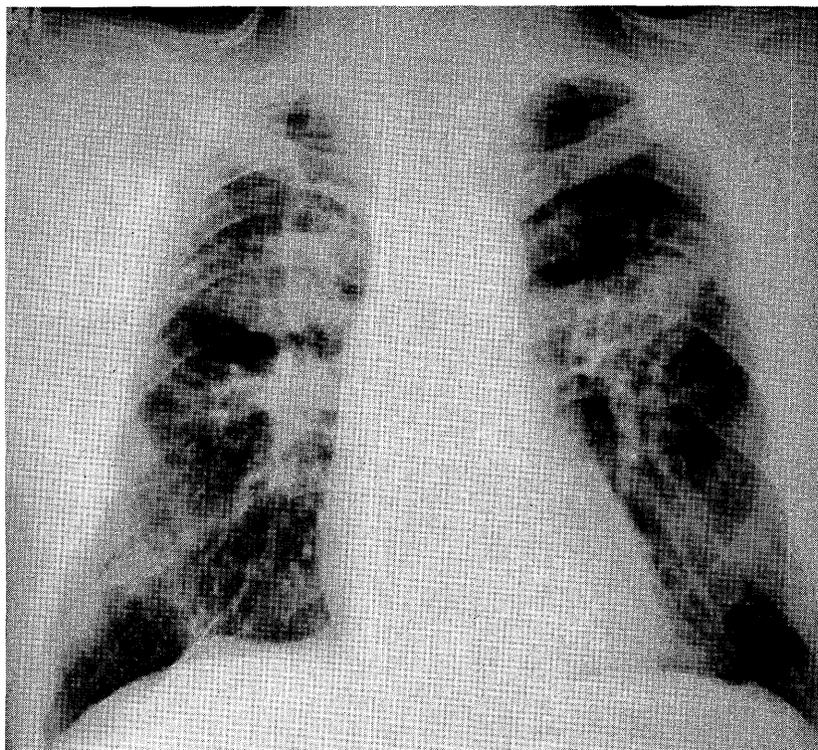


FIGURE 3. Chest radiograph of subject with complicated pneumoconiosis (progressive massive fibrosis).

coniosis as being of little consequence other than as the first indication of dust retention.

How then can the lower ventilatory capacity of coal miners be explained? There are several possibilities: first, that the inhalation of coal dust produces a form of obstructive airway disease or industrial bronchitis. There is some evidence to suggest that this is indeed so, however, the effects of coal dust are so over-shadowed by the effects of cigarette smoking, that it is difficult to confirm this hypothesis. Moreover, whatever the cause of bronchitis, be it cigarette smoking or dust inhalation, the symptoms and physiological abnormalities are the same. Muddying the water more is the fact that miners' wives have more respiratory symptoms, eg, cough and sputum, than do the wives of non-miners. A second possibility is related to the smoking habits of the coal mining population. The average coal miner smokes as many cigarettes in a day as does the non-miner, however, since

he is unable to smoke while at work, he must get through them in a much shorter period. A further possible explanation might be found in differential migration. Were the fitter miner to move out of coal mining after a few years, then the less healthy would remain. This would account for the lower pulmonary function in those remaining. Suffice it to say that whatever the cause of the lower ventilatory capacity in coal miners, it still remains a matter of speculation.

Thus it is evident that as far as simple pneumoconiosis is concerned the diagnosis depends entirely on radiological criteria. There are neither specific symptoms nor characteristic abnormalities of pulmonary function that enable the physician to say the patient has the disease. Finally, it is generally accepted that simple pneumoconiosis is not associated with much in the way of respiratory disability, although most authorities agree that subjects with category 3 simple pneumoconiosis have

a slightly lower ventilatory capacity than do subjects with categories 1 and 2.

Progressive Massive Fibrosis (PMF)

Certain subjects with simple pneumoconiosis go on to develop progressive massive fibrosis. This serious complication occurs in around one to three per cent of active miners. It is seldom seen except on a background of either category 2 or 3 simple pneumoconiosis. While it is in part related to the duration of exposure to coal dust and is almost never found in miners who have not worked for at least 15 years underground, other factors undoubtedly play a role in its pathogenesis, viz., tuberculosis in some instances, non-specific infections in others, and possible autoimmunity in all. Progressive massive fibrosis appears on a chest film as a dense opacity or opacities than initially may be relatively small, but may grow to occupy almost the entire lung. Histological examination reveals large areas of collagenous fibrosis with frequent cavitation. The latter may be a consequence of either ischemic necrosis or tuberculosis. In long-standing cases, when the densities are large, there is a reduction of the alveolo-capillary surface and the pulmonary capillary blood flow is compromised. This leads to pulmonary hypertension and cor pulmonale.

In most subjects with progressive massive fibrosis there is quite marked respiratory impairment. The majority show a reduction in their vital capacity and often in their other lung volumes as well. A lowering of the arterial pO_2 tension is almost always found, this usually being a consequence of ventilation-perfusion abnormalities. Some degree of obstruction may be present. The diffusing capacity may be markedly reduced and the magnitude of the reduction is closely related to the size of the conglomerate masses. Pulmonary hypertension is common. Thus any subject who presents with the radiological features of progressive massive fibrosis, and who is obviously disabled owing to respiratory impairment, can legitimately be assumed to have acquired his disability as a consequence of his occupation.

In conclusion, it can be said that coal

workers' pneumoconiosis is a disease produced by the inhalation of coal dust. Without coal other factors play a role in its development and in this connection some elaboration is necessary. The British have shown that in general coal workers' pneumoconiosis occurs more commonly in mines with high levels of respirable dust, yet there have been, and still are, obvious exceptions to this rule. They therefore considered what other factors might influence the frequency of the disease. The silica content of the coal mined seemed to have little or no effect, but when they looked at the rank (grade) of the coal mined, they found a relationship between it and the prevalence of the disease, viz., the higher the rank, the greater the prevalence. A similar situation exists in the United States.⁷ The rank of a particular coal is related to its age, its carbon content, its volatility, and to the number of British thermal units it liberates per unit weight when burnt. In general, higher ranked coals produce more heat, contain less volatile matter and have a higher carbon content. Another factor which probably influences the prevalence of the disease is the volume of air the miner breathes at work; tasks which involve hard physical work necessitate a higher minute volume and this is likely to lead to more dust being deposited in the alveoli. With increasing mechanization, prolonged physical exertion is becoming increasingly uncommon. Lastly, the rate of clearance from the respiratory tract of inhaled particles varies from one person to another and there is little doubt that this is a partial explanation of why some miners develop the disease while others do not. In this context, the deleterious effects of smoking on ciliary function would seem to place cigarette smoking miners in double jeopardy.

No matter how one looks at the problem, and it is a problem of some magnitude, the answer must lie in dust control. The British have shown what can be done with efficient dust control; there is every reason to try and emulate them, always remembering that the American miner produces five tons of coal for every one that his British counterpart produces and that absolute parity, viz., in coal

production and dust levels, would mean that the United States coal industry would be out of business in a month.

References

1. COLLIS, E. L., and J. C. GILCHRIST: Effects of Dust on Coal Trimmers. *J. Ind. Hyg.* 10: 101 (1928).
2. GOUCHER, J.: Pneumoconiosis in Coal Trimmers. *J. Path. Bact.* 51: 277 (1940).
3. WATSON, A. J., J. BLACK, A. T. DOIG, and G. NAGEL-SCHMIDT: Pneumoconiosis in Carbon Electrode Makers. *Brit. J. Ind. Med.* 16: 274 (1959).
4. GILSON, J. C., and P. HUGH-JONES: *Lung Function in Coal Workers' Pneumoconiosis*. Medical Research Council Special Report Service. Her Majesty's Stationery Office, London, 290 (1955).
5. BRASSEUR, L.: *L'Exploration Fonctionnelle Pulmonaire dans la Pneumokonoise des Houilleurs*. Editions Arscia, Brussels (1963).
6. LYONS, J. P., W. G. CLARKE, A. M. HALL, and J. E. COTES: Transfer Factor of the Lung in Simple Pneumoconiosis of Coal Workers. *Brit. Med. J.* 4: 772 (1968).
7. MORGAN, W. K. C.: Prevalence of Coal Workers' Pneumoconiosis. *Amer. Rev. Resp. Dis.* 98: 306 (1968).

Received May 14, 1970

Information Costs

Effective January 1, 1971, the National Technical Information Service increased the prices for document copies and microfiche in certain categories.

Paper copy (hard copy) of most documents announced after January 1, 1969 are priced:

1 to 300 pages	\$3.00
301 to 600 pages	6.00
601 to 900 pages	9.00
Over 900 pages	Exception Price

A plus sign in lieu of a price in announcement of a document indicates that the material is available upon request. Two years after announcement, documents having 300 pages or less will have a service charge of \$3.00 added to the announcement price. No service charge will be added for documents over 300 pages. Documents announced prior to January 1, 1969, have a service charge of \$3.00 added to the announcement price.

Microfiche copies of documents on a demand basis are priced at 95 cents per document. Documents available on Standing Order through NTIS Selective Dissemination of Microfiche Service are priced at 35 cents per document.

Further information concerning the prices and concerning the services of the National Technical Information Service may be had by writing to them at 5285 Port Royal Road, Springfield, Virginia 22151.

Course on Use of Enzymes

The Massachusetts Institute of Technology Summer School program will present a course "Enzymes and Their Use in Analysis and Clinical Diagnosis" at Cambridge, Massachusetts, July 12-July 16, 1971. The objective of the course is to prepare the participants for the use of enzymes as analytical reagents and for the measurement of enzyme activities in biological materials. A comprehensive theoretical background will be presented covering enzyme synthesis and its regulation, enzyme purification, kinetics of enzyme catalyzed reactions, distribution of enzymes, and isoenzymes in tissues. The methodology for enzyme assays will be considered in detail and illustrated by various demonstrations in the laboratory. For further information on this course write to: Director of the Summer Session, Room E19-356, Massachusetts Institute of Technology, Cambridge, Massachusetts 02139.