RESPIRATORY IMPAIRMENT IN WORKING COAL MINERS

by

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INTRODUCTION

It is fairly widely accepted that coal miners have a greater prevalence of respiratory disease than does the general population (1-2, 4).[2] The extent and cause of the excess of respiratory disease are still the subject of debate, but there seems little doubt that the geographical region in which the miner works has a bearing on the prevalence of respiratory symptoms and impairment. There are several factors that must be considered in the etiology of respiratory disease in coal miners. First, there are those related to his occupation per se, namely, coal mine dust and possibly other noxious agents to which he is exposed while at work. Secondly, there are the factors that are responsible for the development of naturally occurring respiratory diseases, such as, chronic obstructive airway disease, tuberculosis, and lung cancer, all of which also affect the general population and are clearly not work related.

Coal workers' pneumoconiosis (CWP) is a consequence of exposure to dust and there is little doubt that in its complicated form, it is associated with respiratory disability and premature death (3, 9). In contrast, simple CWP is associated with some minor respiratory impairments, which of themselves are not severe enough to be associated with respiratory disability (6). It is accepted by informed physicians that although the higher grades of simple pneumoconiosis (categories 2 and 3) produce little in the way of respiratory disability, there is an increased risk of miners with these categories developing the complicated form of the disease. Thus, simple pneumoconiosis, although relatively harmless by itself, is sometimes a precursor of serious disease, namely complicated pneumoconiosis.

There is some debate as to whether chronic nonspecific obstructive airway disease occurs more frequently in coal miners and, as already mentioned, investigations into the prevalence of airway obstruction and bronchitis in miners have produced contradictory results. Several Appalachian Laboratory for Occupational Respiratory Diseases (ALFORD) studies have a bearing on this problem and are worth description.

It is proposed in the remainder of this paper to describe the types of physiological respiratory impairment that are seen in coal miners, and to attempt to decide which of these impairments are related to coal mining and which to naturally occurring disease.

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[2]Underlined numbers in parentheses refer to items in the list of references at the end of this paper.

VENTILATORY CAPACITY

Most studies in Britain and the United States have shown that coal miners have a lower ventilatory capacity than that found in comparable groups of non-miners (1-2). In some studies, differences have been quite obvious and in others less so. Thus, Enterline was able to show a difference between coal miners and a comparable group of railway workers when he surveyed a random population in Mullens, W. Va. On the other hand, in Richwood, W. Va., his findings were equivocal and there was no real difference in the ventilatory capacity of the Richwood miners from that of the control population of lumber men (2). Preliminary analysis of the data that we have collected in the Interagency Study of Coal Workers' Pneumoconiosis shows similar inconsistencies. There is little doubt that anthracite miners have a lower ventilatory capacity than do either their bituminous counterparts or the normal population. Thus, their forced expiratory volume in 1 second (FEV_1), forced vital capacity (FVC), and residual volume (RV) all deviate significantly from the predicted figure and the extent of the deviation is related to their category of simple pneumoconiosis. In addition, anthracite miners have more bronchitis and more chest symptoms than do their bituminous colleagues and the excess of symptoms cannot be accounted for by their smoking habits (8). In the case of bituminous miners, those who come from central Pennsylvania, southern West Virginia, Virginia, eastern Kentucky, and Alabama seem to have an excess of respiratory symptoms but only a slight reduction of ventilatory capacity. Lacking a comparable control group, their mean spirometric values have been compared with the predicted figures of Kory and others (5). In general, they are slightly but significantly lower. In contrast, the comparable figures for miners from the Middle and Far West compare very favorably with the predicted figures and in some instances are significantly better. This is especially true in Utah and Colorado. Furthermore, there seems to be no obvious relationship between the extent of the ventilatory impairment and the category of simple pneumoconiosis except in the anthracite miners. As is to be expected, definite ventilatory impairment was present in progressive massive fibrosis (PMF). There seems therefore to be some evidence that in certain regions, there is a reduction of the ventilatory capacity of nonpneumoconiotic working miners over and above that produced by naturally occurring obstructive disease. The impairment that is a consequence of their occupation is not of sufficient severity to be associated with disability with the exception of that occurring in the anthracite miners from eastern Pennsylvania.

LUNG VOLUMES

Previous studies carried out in this laboratory have shown that the residual volume of the lungs of working miners is increased and that the extent of the increase is related to the radiographic category (7). This applies whether or not the miners have evidence of obstructive airway disease, and is present in nonsmokers. We feel that the increased residual volume is a consequence of obstruction in the small airways rather than focal emphysema.

DIFFUSING CAPACITY

The problems of measuring the diffusing capacity in a large group of subjects are those concerned with selection. In particular, there is the problem of excluding the effects of coincident, naturally occurring, obstructive airway disease. Similarly, it has been shown that cigarette smokers have small but significant abnormalities of their diffusing capacity and in order, therefore, to study the effects of coal workers' pneumoconiosis on the diffusing capacity, it is necessary to select a group of miners who are nonsmokers, who have no evidence of obstruction of the large airways, and who have no evidence of concomitant cardiopulmonary disease other than CWP. In a group of 30 nonsmoking miners with categories of 2 and 3 simple pneumoconiosis, we showed that none of them had a significant reduction of their diffusing capacity (10). There was a minor reduction in the mean values for diffusing capacity in the miners with the pinhead type of opacity as compared to those who had micronodular opacities. This reduction, however, was small and only significant when the two groups were considered as a whole.

DISTRIBUTION OF INSPIRED GAS

Previous studies carried out at the Appalachian Laboratory for Occupational Respiratory Disease (ALFORD), have shown that working and ex-coal miners with pneumoconiosis have minor abnormalities in the distribution of inspired gas (6). These consist of changes in the ratio of dead space to total volume and in the alveolar-arterial gradient for oxygen. In no instance were these abnormalities sufficiently large to be associated with disability.

ARTERIAL BLOODS

Simple pneumoconiosis by itself does not produce significant desaturation, although the latter is frequently present in the complicated form of the disease (6).

MECHANICS OF RESPIRATION

Past investigations carried out in ALFORD have shown that nonsmoking miners with simple pneumoconiosis demonstrate a fall in their compliance at high rates of breathing (9). Although this implies that there is an increased resistance to flow in the smaller airways, this finding cannot necessarily be equated with the presence of disability. Studies of compliance and the retractive forces of the lung have shown that they are usually little affected in simple pneumoconiosis. There is a tendency for some subjects to have a slight reduction in the retractive forces; a finding that can be attributed to the presence of focal emphysema (PMF). In PMF, especially of the more advanced stages, the lungs are much less distensible than normal, and the retractive forces are increased. The cardiopulmonary laboratory of ALFORD has also shown that the flow rates of miners with pneumoconiosis are somewhat reduced and the reduction is not always a consequence of low retractive forces. This finding is again compatible with the presence of small airway disease.

NONSPECIFIC OBSTRUCTIVE AIRWAY DISEASE

Up to now the various impairments that have been described are a consequence of CWP and can be clearly related to the inhalation of coal dust particles of the respirable range. As mentioned earlier, there is some evidence that nonspecific obstructive airway disease occurs more frequently in coal miners. While there is little doubt that cigarette smoking is preeminent in the etiology of this entity, nonetheless, studies of the prevalence of bronchitis in working miners have shown that in nonsmokers there is clearcut relationship between bronchitis and dust exposure. Thus, nonsmoking, face workers have a greater prevalence of bronchitis and a slightly lower ventilatory capacity than do their counterparts who work on the surface. In smokers, the effects of dust exposure are overwhelmed by the effects of cigarette smoking and no discernible trend is evident. Furthermore, the presence of bronchitis does not seem to be related to radiographic evidence of coal workers' pneumoconiosis. There is good reason to assume that the type of dust which is responsible for the development of bronchitis differs from that which is responsible for the production of CWP. Bronchitis by definition implies a disorder of the mucous glands and goblet cells present in the conducting system of airways. Few particles in the respirable range are deposited in these airways, and it seems that dust induced bronchitis is more likely to be an effect of the deposition of larger particles in the conducting system, namely, those between 5 and 15 microns. Unfortunately, the Bureau of Mines is not making total dust measurements and is confining itself to making measurements of dust particles in the respirable range. It would seem desirable to correct this omission.

In addition to the chronic effects of dust exposure, studies carried out by Dr. Lapp and his colleagues have shown that there is an acute effect of coal dust exposure. In a study carried out by ALFORD personnel, it was shown that the ventilatory capacity of miners declined following a work shift. Further experiments using similar techniques and which relate changes in function to prevailing dust levels are desirable.

Although much is understood concerning the development and effects of pneumoconiosis and other respiratory impairments in coal miners, much still remains to be done in this field. The effect of coal dust on the smaller conducting airways is only just starting to be investigated and there is room for great expansion in this field. Similarly, it is becoming apparent that both the prevalence of pneumoconiosis and the prevalence of respiratory impairment differ in various geographical regions. At present no real explanation for these differences exist. This may be a consequence of physical or chemical composition of coal dust to which the men are exposed, and appropriate studies to unravel these problems could profitably be carried out by the Bureau of Mines.

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Information Circular 8568

Coal Mine Health Seminar

A Joint Staff Conference of the Bureau of Mines and the National Institute for Occupational Safety and Health

Compiled by Marilyn K. Hutchison, M.D.

UNITED STATES DEPARTMENT OF THE INTERIOR
Rogers C. B. Morton, Secretary

BUREAU OF MINES
Elburt F. Osborn, Director

This publication has been cataloged as follows:

U.S. Bureau of Mines

Coal Mine Health Seminar: A Joint Staff Conference of the Bureau of Mines and the National Institute for Occupational Safety and Health [Sept. 6, 1972. Washington] U.S. Dept. of the Interior, Bureau of Mines [1972]

44 p. illus., tables. (U.S. Bureau of Mines. Information circular 8568)

1. Coal mines and mining–Safety measures. 2. Coal mines and mining–Congresses. I. U.S. National Institute for Occupational Safety and Health. II. Title. (Series)

TN23.U71 no. 8568 622.06173

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