


281


CHAPTER 7

RESPIRATORY DISEASE
IN COAL MINERS
CONTENTS

Introduction

Prevalence of Smoking in Coal Miners

Coal Workers' Pneumoconiosis
  Prevalence of Coal Workers' Pneumoconiosis
  Pulmonary Function Abnormalities in Simple and Complicated Coal Workers' Pneumoconiosis
  Relationship of Small Opacities to Emphysema and Airways Obstruction
    Lung Function in Subjects With Rounded or Regular Opacities
    Lung Function in Subjects With Irregular Opacities

Respiratory Symptoms and Exposure to Coal Dust
  Bronchitis and Dust Exposure

Respiratory Mortality in Coal Miners

Lung Function in Coal Miners
  Emphysema, Exposure to Coal Dust, and Cigarette Smoking
  Dust Exposure, Cigarette Smoking, and Ventilatory Function

Summary and Conclusions

References
Introduction

An association between respiratory disease and coal mining has been recognized since the 16th century, when Agricola and Paracelsus wrote of the diseases of miners (Hunter 1978). The first description of coal workers' pneumoconiosis (CWP) was given in the early 1800s by Laennec (Meiklejohn 1951) when he described cystic and noncystic melanotic masses in the lung, and in addition, melanotic parenchymal infiltrates in the lung. The melanotic masses were almost certainly progressive massive fibrosis (PMF) and the black infiltrates, simple CWP. It is clear from Laennec's description that he recognized an association between coal mining and the deposition of "la matiere noire pulmonaire." An excellent history of coal miners' lung disease in Great Britain can be found in a series of articles by Meiklejohn (1951, 1952a, 1952b).

Over the years, a large number of names have been attached to the conditions that affect the lungs of coal miners. Many early physicians assumed that there was a single respiratory condition arising from coal dust exposure, which was variously referred to as spurious melanosis, miners' asthma, anthracosis, miners' phthisis, and silicosis. With the passage of time, it became evident that, aside from the lung diseases that commonly affect the general population, coal miners are prone to develop occupationally related lung diseases, namely coal workers' pneumoconiosis, silicosis, and chronic bronchitis (Morgan and Lapp 1976). Silicosis is covered elsewhere in this Report; therefore this chapter discusses coal workers' pneumoconiosis and chronic bronchitis.

The paramount importance of exposure to coal dust in the development of CWP is generally accepted, and complicated CWP is clearly associated with significant and often disabling chronic airflow limitation as well as with other respiratory impairments (Morgan and Seaton 1984; Morgan and Lapp 1976). Less certain is the magnitude of the role of coal mine dust as a cause or contributory factor in the development of bronchitis and emphysema. The effects of long-continued inhalation of coal and other dusts are currently of major interest to epidemiologists and to those practicing occupational medicine. Clearly the results of the studies designed to characterize the effects of coal dust on lung function are of vital importance to officials concerned with compensation for occupationally related pulmonary disability. In these studies it is important to evaluate as potentially independent effects the role of coal dust in producing radiologic CWP and the role of coal dust in producing physiologic airflow obstruction (Weeks and Wagner 1986). This separation of the radiologic and physiologic responses to coal dust is even more critical when considering the effects of combined exposure to coal dust and cigarette smoke.
The respective contributions of cigarette smoking, dust exposure, and other environmental and occupational factors in the development of respiratory impairment in coal miners are examined in this chapter by reviewing the evidence currently available from mortality and morbidity studies of coal miners compared with appropriate reference populations and the evidence on the frequency and extent of pulmonary impairment in coal miners. The roles of dust, cigarette smoking, and various confounding factors are taken into account and apportioned where possible.

At the present time, there are approximately 150,000 underground coal miners in the United States. Ten years ago, the figure was about 170,000, but the closure of a number of mines in Appalachia has reduced the number of employed underground miners. In addition, 60,000 to 70,000 workers are employed in open cast or surface mines, but this number is constantly changing. Exposure to coal mine dust is greater in those employed underground; miners working at the face or in transportation are the most heavily exposed. Miners employed underground on maintenance and other activities are less exposed, and surface miners are the least exposed (Doyle 1970).

Prevalence of Smoking in Coal Miners

The prevalence of smoking for various populations of miners in different countries and during different time periods is presented in Table 1. In general, the prevalence of smoking among U.S. coal miners is currently similar to, or slightly higher than, the rates in the overall U.S. male population. However, coal mining as an occupation introduces a distortion in the pattern of smoking because of the prohibition against smoking while in the mine. As a result, the entire consumption of cigarettes by miners is limited to those periods when they are not underground; for a given number of cigarettes smoked per day, the pattern among miners would consist of periods of more intense smoking interspersed with long periods of not smoking (i.e., during working hours), in contrast to the more even consumption of cigarettes throughout the day that characterizes most cigarette smokers.

Coal Workers' Pneumoconiosis

Coal workers' pneumoconiosis (CWP) is defined as the deposition of coal mine dust in the lungs and the reaction of tissue to its presence (Morgan and Seaton 1984). However, the term is commonly applied only to the chest roentgenographic changes produced by coal dust, and the other tissue responses to coal dust are classified by their symptomatic, physiologic, or pathologic manifestations (e.g., chronic bronchitis, airflow obstruction, or emphysema). The radiologic
TABLE 1.—Smoking characteristics of coal workers

<table>
<thead>
<tr>
<th>Study</th>
<th>Number and type of population</th>
<th>Smoking characteristics (percent)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Higgins et al. (1968)</td>
<td>766 mining town residents, Great Britain</td>
<td>SM 75 NS 25</td>
<td></td>
</tr>
<tr>
<td>Tokuhata et al. (1970)</td>
<td>801 anthracite coal miners, Pennsylvania</td>
<td>Large collieries Age &lt; 40 SM 84 1 NS 15.9 EX 11.9 Age 40-49 SM 74 0 NS 26.0 Age &gt; 50 SM 71 6 NS 28.4 Independent Age &lt; 40 SM 76 4 NS 23.6 Age 40-49 SM 80 5 NS 19.5 Age &gt; 50 SM 77 3 NS 22.7</td>
<td></td>
</tr>
<tr>
<td>Ashford et al. (1970)</td>
<td>30,000 underground and surface workers, Great Britain</td>
<td>SM 78 7 NS 15.3 EX 6</td>
<td></td>
</tr>
<tr>
<td>Rae et al. (1971)</td>
<td>3,379 workers, 20 collieries, Great Britain</td>
<td>SM 79 2 NS 20.8</td>
<td>Ex-smokers not differentiated from smokers</td>
</tr>
<tr>
<td>Stasic and Falcic (1971)</td>
<td>Lignite and brown coal miners, Yugoslavia</td>
<td>SM 64 7 NS 23.6 EX 11.7</td>
<td></td>
</tr>
<tr>
<td>Study</td>
<td>Number and type of population</td>
<td>Smoking characteristics (percent)</td>
<td>Comments</td>
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<tr>
<td>Minette (1972)</td>
<td>204 coal miners, Belgium</td>
<td>SM 70.6 NS* 29.4</td>
<td>*Non-smokers include smokers who did not inhale</td>
</tr>
<tr>
<td>Phelps (1972)</td>
<td>256 miners, U.S. Rocky Mountain region</td>
<td>SM 65.6 NS 34.3</td>
<td></td>
</tr>
<tr>
<td>Skrabski-Kopp et al. (1972)</td>
<td>1,068 coal miners, Hungary</td>
<td>SM 73.6 NS/EX 24.4</td>
<td></td>
</tr>
<tr>
<td>Lowe and Khosla (1972)</td>
<td>9,012 ex-coal miners, 8,361 controls, Great Britain</td>
<td>Ex-miners 86.6 Controls 80.7</td>
<td></td>
</tr>
<tr>
<td>Kiblstie et al. (1973)</td>
<td>Bituminous coal miners, United States</td>
<td>SM 50.8 NS 25.4 EX 23.8 90% age-old miners smoked at a rate above 65%</td>
<td></td>
</tr>
<tr>
<td>Orteneyer et al. (1974)</td>
<td>Coal miners and ex-miners, Appalachia</td>
<td>Coal miners 60.9 Ex-miners 47.1 19.7 19.4 52.3</td>
<td></td>
</tr>
<tr>
<td>Study</td>
<td>Number and type of population</td>
<td>Smoking characteristics (percent)</td>
<td>Comments</td>
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<tr>
<td>Armstrong et al.</td>
<td>Coal and gold miners, Australia</td>
<td>SM 58.1, NS/EX 41.3</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Gold miners 66.3</td>
<td></td>
</tr>
<tr>
<td>Szymczykiewicz</td>
<td>Miners, Poland</td>
<td>73.7</td>
<td></td>
</tr>
<tr>
<td>Potkonjak</td>
<td>Coal miners, 970</td>
<td>Miners 40, Controls 47.9</td>
<td></td>
</tr>
<tr>
<td></td>
<td>controls with no dust exposure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cochrane and Moore</td>
<td>Coal miners, Great Britain</td>
<td>Age 55-64: SM 24.3, NS/EX 47.9,  Pipe only</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Age &lt;55: SM 71.7, NS/EX 24.3,</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>1-14: 32, &gt;14: 39.3</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Age 55-64: SM 23.6, NS/EX 47.9,</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>1-14: 32, &gt;14: 28</td>
<td></td>
</tr>
<tr>
<td>Rom et al.</td>
<td>242 active and retired miners, Utah</td>
<td>SM 38, NS 23.6, EX 38.4</td>
<td></td>
</tr>
<tr>
<td>Love and Miller</td>
<td>1,677 colliery workers, Great Britain</td>
<td>SM 66.1, NS 12.5, EX 4.5</td>
<td></td>
</tr>
</tbody>
</table>

NOTE: SM = Smoker, EX = Ex-smoker, NS = Nonsmoker.
manifestations of CWP are classified into two forms: simple, and complicated (often known as progressive massive fibrosis, PMF). Simple CWP is a reaction solely to the inhalation of coal dust. Although the efficiency and integrity of the lung defenses are important factors in the development of disease, cumulative dust exposure is of paramount importance. Characteristically, after a period of exposure, i.e., 10 to 15 years, small rounded opacities begin to appear in the upper lung fields of some miners. With continued exposure, they gradually spread to the middle and lower zones, and the increasing profusion of these opacities is used to categorize simple CWP on a 0 to 3 scale. The opacities are indistinguishable from those seen in silicosis. Simple CWP does not progress in the absence of further exposure. Moreover, increasing category of simple CWP is not associated with a decrement in ventilatory capacity (Cochrane et al. 1961; Gilson and Hugh-Jones 1955; Morgan et al. 1974).

Complicated CWP is defined as the presence of an opacity greater than 1 cm in diameter on the chest radiograph of a subject who already has simple CWP (ILO 1980), and the volume of lung occupied by the large opacities on the radiograph is used to categorize complicated CWP into category A, B, or C. For the most part, complicated CWP develops on a background of category 2 or category 3 simple CWP. Necessary for its development is the presence of a fairly high dust burden in the lungs, plus some other factor or factors as yet not fully recognized or understood.

**Prevalence of Coal Workers’ Pneumoconiosis**

In 1969 the National Coal Study was commenced by the National Institute for Occupational Safety and Health (NIOSH) (Morgan et al. 1972). Selected for study were workers at 31 coal mines, of which 2 were anthracite and 29 were bituminous. In the initial survey, an overall CWP prevalence of nearly 30 percent was found; 2.5 percent had PMF. A decreasing prevalence was noted from east to west, and there was a clear-cut relationship between the years spent underground and radiographic category. The disease was much more common in workers at the face than in surface workers. The prevalence of CWP in this study was undoubtedly overestimated, however. Part of the reason was that the early films were interpreted according to the 1958 International Labour Office (ILO) classification. In addition, some of the readers were inexperienced and tended to overread. The second round of the National Coal Study revealed a prevalence of 8 percent; the findings at the third round were just under 5 percent, with 3.85 percent having category 1; 0.48 percent, category 2; 0.04 percent, category 3; and 0.17 percent, PMF.

The decline in the prevalence of pneumoconiosis noted between the first and third rounds of the National Coal Study is partly
accounted for by the use of different readers, partly by more stringent reading criteria, partly from the use of the ILO standard films as yardsticks, and partly from improved coal dust control. An exodus of workers with higher categories of disease between the first and subsequent accumulations of subjects with radiographic evidence of pneumoconiosis also played a role, since those with CWP qualified for compensation.

Both simple CWP and PMF are related to cumulative lifetime exposure to coal dust. The reduction or elimination of category 2 and category 3 simple CWP through effective dust control not only is possible but is rapidly being achieved in the United States and Great Britain (Jacobsen 1980). Since complicated pneumoconiosis develops almost entirely in subjects who have the higher categories of simple CWP (i.e., those with a high dust burden), the effective prevention of category 2 and category 3 simple CWP should almost completely eliminate PMF as well. The removal from further coal dust exposure of miners with early categories of simple CWP should also aid in reducing the incidence of PMF.

Pulmonary Function Abnormalities in Simple and Complicated Coal Workers' Pneumoconiosis

The radiologic changes of simple CWP are associated with the development of certain relatively minor pulmonary impairments. These include an increased alveolar-arterial gradient for oxygen, abnormalities of the distribution of inspired gas, and a modest increase in lung volumes (Morgan and Lapp 1976). The increase in lung volumes is a consequence of the focal emphysema that is an integral part of the pathology found with simple CWP (Morgan and Seaton 1984).

Complicated pneumoconiosis is associated with a reduction in lung volume and diffusing capacity, ventilation perfusion mismatching, an obliteration and destruction of the pulmonary vascular bed that leads to nonhypoxic pulmonary hypertension and cor pulmonale, and with the presence of generalized airways obstruction (Gilson and Hugh-Jones and Seaton 1955; Lyons et al. and Seaton 1981; Morgan and Seaton 1984).

Relationship of Small Opacities to Emphysema and Airways Obstruction

The small opacities present in the lung in the various pneumoconioses can be classified as either rounded (regular) or irregular.

Small rounded opacities have a fairly rounded and regular margin. They are classified according to their size into p, q, and r types: p is up to 1.5 mm in diameter, q is between 1.5 and 3 mm, and r is between 3 and 10 mm. Usually only one type of opacity is present,
but mixtures are occasionally found in the same lung. The reading of
the film should be based on the predominant opacity noted.

Irregular opacities are also classified according to size: s opacities
are up to 1.5 mm in width, t opacities are between 1.5 and 3 mm
wide, and u opacities are between 3 and 10 mm. Irregular opacities
are characteristically seen in asbestosis. In some smokers scanty
irregular opacities may be observed.

Rounded and irregular opacities occasionally occur together,
usually in a person who has experienced various exposures or in a
person with either silicosis or coal workers' pneumoconiosis who also
is a heavy smoker.

Lung Function in Subjects With Rounded or Regular Opacities

A number of investigations have shown that the p, or punctate,
type of opacity seen in simple CWP is associated with a reduced
diffusing capacity (Sartorelli et al. 1963; Lyons et al. 1967; Seaton et
al. 1972; Musk et al. 1981). In addition, an increased parenchymal air
space size has also been observed in subjects with a p type of opacity
(Hankinson et al. 1979). No detectable difference in lung function
between subjects with the p, q, or r type of opacity was observed in
this study.

These physiological changes have also been demonstrated in
nonsmoking miners with simple CWP (Hankinson et al. 1979; Seaton
et al. 1972). The indices of lung function that were tested in the
miners with the p and q opacities were similar (Hankinson et al.
1979) except for the diffusing capacity, which was significantly lower
in those with the p type of lesion (Seaton et al. 1972). Static
compliance was reduced marginally, but not significantly, with the q
type of opacity. The type ofopacity was not significantly related to
differences in dynamic compliance at increased rates of breathing
(Seaton et al. 1972).

Musk and colleagues (1981) examined the lung function in 125 coal
miners identified in 1968 as having the simple pneumoconiosis of
coal workers and reexamined 9 years later. Pulmonary function was
related to both smoking history and coal dust exposure. Miners who
smoked in 1978 had lower forced expiratory volume in 1 second
( FEV1), forced vital capacity (FVC), and FEV1/FVC ratio and a
higher ratio of residual volume to total lung capacity (RV/TLC)
compared with nonsmokers. Ex-smokers had a lower carbon monox-
ide diffusing capacity (DLCO) than nonsmokers. Total dust exposure
was inversely related to FVC and lung elastic recoil at TLC. After
correcting for the effects of age, height, and smoking category,
miners whose radiographs showed predominantly p and r types of
opacities had a reduced DLCO when compared with miners with the
q type of opacity. In the researchers' experience, irregular opacities
were also associated with a reduced DLCO, and were thought to
reflect the presence of both emphysema and diffuse fibrosis. In addition, the r type of opacity, but not the p type, was associated with reduced maximal recoil at TLC, and reduced recoil at 50 percent of TLC and at 1 L below TLC, and with an increased RV and RV/TLC percent compared with miners with the q type of opacity. The decreased compliance in subjects with the r type of opacities noted by Musk and colleagues (1981) may be a consequence of the exposure of some of these subjects to silica. Irregular opacities were not associated with increased obstruction or with smoking; here the results of Musk and colleagues differ from almost all other studies.

Lung Function in Subjects With Irregular Opacities

The significance of irregular opacities in the lungs of coal miners has been the subject of a number of investigations. Lyons and colleagues (1974) pointed out that there was a positive association between the presence of irregular opacities and emphysema and impairment of FEV₁. Unfortunately, this was a post-mortem study in which no smoking histories were available. Subsequently, Amandaus and colleagues (1976) investigated the significance of irregular opacities in the lungs of 6,166 working U.S. coal miners for whom complete smoking histories were available. Irregular opacities on the chest radiographs were shown to be associated with smoking, bronchitis, age, and years worked underground. Smoking was not associated with the presence of regular opacities. Although irregular opacities were observed in nonsmokers, they were 2.5 times more common in those who smoked. Among nonsmoking miners, there were no significant differences in the lung volumes or flow rates of the men with normal chest x rays, irregular opacities, rounded opacities, or mixed opacities. Smokers had similar FVCs in each of the radiologic categories when compared with nonsmokers, but FEV₁ and FEV₁/FVC were lower and RV and TLC were higher. In addition, smokers with irregular opacities had a lower FEV₁ and a higher RV and TLC than smokers with normal chest x rays or rounded opacities. A study by Cockcroft, Berry, and colleagues (1982) of coal workers and ex-workers showed that irregular opacities were related to age, smoking, and underground exposure in those receiving disability benefits.

Cockcroft, Seal, and colleagues (1982) examined the relationships among lung function tests, irregular opacities on chest radiograph, and the pathologic changes of emphysema in 46 men who had been referred for lung function tests during life and who had died between 1970 and 1979. Irregular opacities on radiograph were associated with a reduced DLCO and reduced TLC, an increased pathologic score for emphysema, and to a lesser extent, an increased pathologic score for fibrosis. Smoking histories were obtained in all but five of these workers, and there was no association of smoking with any
particular lung function or pathologic finding. However, almost all of the subjects in the study were current smokers (two were nonsmokers and two were former smokers), which limited the ability of the study to examine the effects of smoking.

The majority of the evidence indicates that simple CWP is associated with mild overdistension or hyperinflation of the lungs. There is some evidence, especially in miners with the p type of opacity, that there is also a reduction in DLCO. The decreased DLCO does not appear to be associated with increasing airways obstruction, but with focal dust emphysema. The available data indicate that cigarette smoking plays a much greater role in reducing DLCO than does the presence of simple CWP (Frans et al. 1975).

Irregular opacities occur occasionally in the lungs of coal miners and former miners. For the most part, they are associated with smoking, age, bronchitis, and years spent underground. Bronchitis may be the common denominator in the production of irregular opacities, and the increased prevalence of bronchitis in smoking coal miners may be the reason for the increased prevalence of irregular opacities found among smokers in some studies. Irregular opacities are seen in nonoccupationally exposed groups (Carilli et al. 1973), in subjects exposed to silica, in asbestos miners and millers (Morgan 1978), in workers who manufacture manmade fibers (Weill et al. 1983), and in workers with other conditions, suggesting that irregular opacities may be a nonspecific response associated with the presence of bronchitis, regardless of its etiology.

**Respiratory Symptoms and Exposure to Coal Dust**

The relationship between dust exposure and bronchitis was noted by Thackrah (1832) and Greenhow (1860) in the 19th century. Although these pioneer workers noted a higher prevalence of bronchitis and other respiratory ailments in the dusty trades as a whole, they particularly emphasized the importance of textile dust as a cause of bronchitis.

Until recently the use of the terms "chronic bronchitis" and "emphysema" implied that these two conditions were invariably associated and that both, for the most part, were related to cigarette smoking. In this context, "bronchitis" implied a condition characterized by cough and sputum, usually associated with a reduction in ventilatory capacity or frequently leading to one (Fletcher et al. 1959). Subjects with these symptoms who also had concomitant chronic airflow obstruction were usually diagnosed as having chronic bronchitis and emphysema, assuming from the association of the two diseases that they were part of the same process. At the time the committee appointed by the Medical Research Council (MRC) of Great Britain published its statement (British Medical Journal
1966), it was known that not all subjects with chronic bronchitis showed an associated reduction of FEV₁, but the committee did not elaborate fully on the implications of the term “bronchitis.” It was assumed, moreover, that there was a relationship between the symptoms of cough and sputum and a decreased ventilatory capacity, and that sooner or later most or all subjects with chronic bronchitis would develop some degree of irreversible airways obstruction. The MRC’s (1965) division of bronchitis into obstructive bronchitis and simple bronchitis, a condition characterized by the presence of cough and sputum in the absence of airways obstruction, was the first step taken toward a better understanding of the implications of a diagnosis of bronchitis. Subsequently, the general use of the MRC questionnaire on chronic bronchitis for symptoms without reference to lung function led to an appreciation of the pathophysiology of this condition (MRC 1965), and the pathological features of bronchitis as described by Reid (1960) in her studies provided a means of quantitating the severity of the condition.

**Bronchitis and Dust Exposure**

Many of the studies showing an association between dust exposure and an increased prevalence of chronic bronchitis have been carried out with coal miners. Coal miners represent a clearly defined and relatively large group of subjects who seldom change their occupation, and thus present an ideal study population. Ashford and colleagues (1970) showed that the prevalence of cough and sputum increased with age and, it may be inferred, with cumulative dust exposure. Shortly thereafter, Rae and colleagues (1971) demonstrated a relationship to dust exposure. Kibelstis and colleagues (1973) showed, in a U.S. population of more than 9,000 working coal miners, that cough and sputum were related to dust exposure and also to cigarette smoking. When only nonsmokers were considered, there was a gradient in the prevalence of bronchitis from the least to the most dusty jobs. This occurred independent of age. In smokers, the effect of cigarette smoking almost completely overwhelmed the effects of dust and age, at least as far as symptoms were concerned. Similar findings have been reported in Belgian coal miners (Minette 1976; Vuylsteek and Depoorter 1978).

The effect of dust and cigarette smoke on bronchial gland dimensions in coal miners has recently been investigated (Douglas et al. 1982). These investigators demonstrated that both dust and cigarette smoking had an effect on the Reid index and that they led to mucous gland hypertrophy. There is thus a fairly widespread acceptance that the long-continued inhalation of coal dust and other dusts may lead to an increased prevalence of cough and sputum in the absence of cigarette smoking. Moreover, it has been demonstrated that the prevalence of dust-induced bronchitis is related to
cumulative dust exposure (Rogan et al. 1973; Kibelstis et al. 1973). The topic of industrial bronchitis is more fully discussed in another chapter of this Report, but the data suggest that there is an independent and additive effect of coal dust exposure and cigarette smoking on the prevalence of chronic bronchitis.

Respiratory Mortality in Coal Miners

Early mortality data of coal miners showed a high death rate from respiratory disease. This was true for both Great Britain (Registrar General 1958) and the United States (Enterline 1964; Guralnick 1963). Although there may be some doubt as to the precise accuracy of such data, it is probably true that there was an increased standardized mortality ratio (SMR) for respiratory disease among coal miners. There was, however, little convincing evidence to establish that coal dust was a major causative factor in this increase. Tuberculosis, emphysema, and bronchiectasis were more common in coal miners, and most of the increased mortality could be explained by an increased prevalence of these diseases. In addition, Enterline (1964, 1972) had shown in a series of retrospective analyses that the SMR for coal miners as a group was elevated, but a substantial portion of the excess was a consequence of trauma and accidents. When deaths due to these excesses were excluded, excess mortality still persisted. Much of the excess was due to respiratory disease, and although the death rate for chronic bronchitis and emphysema was reported to be increased, so also were the death rates for tuberculosis and lung cancer. While it is easy to postulate a relationship between occupation and bronchitis, it is also clear that bronchitis and lung cancer have a common causative agent, namely cigarette smoke. In addition, coal miners, particularly in the United States, constituted a distinctly underprivileged group during the early part of this century, and as such suffered from overcrowding and poor medical care, both of which contributed significantly to a higher death rate from bronchiectasis and tuberculosis and other infectious diseases.

Over the past two decades, a number of well-controlled epidemiological studies of morbidity and mortality of coal miners have been carried out in both Great Britain and the United States. Liddell (1973a) looked at the frequency of time off from work because of illness in a cohort of 29,084 men. He showed that miners spent more time off work than nonminers. The highest rate of incapacity was present in the lowest paid workers; this applied as much to coal face miners as to surface workers. Pneumoconiosis was associated with greater time off work. An additional investigation of the mortality of 5,362 British miners who died in 1961 showed that they had higher death rates for accidents and pneumoconiosis than the general population, but lower death rates for cancer in general and for lung...