COAL WORKERS' PNEUMOCONIOSIS AND EXPOSURE TO OTHER CARBONACEOUS DUSTS

James A. Merchant
Geoffrey Taylor
Thomas K. Hodous

INTRODUCTION

Historical accounts of "Miners' Black Lung" date to 1831. Since that time, numerous clinical and epidemiological studies have documented the existence of Coal Workers' Pneumoconiosis (CWP) and associated lung impairment among miners. Extensive British prospective studies have established dose-response relationships between CWP and respirable coal mine dust. The Federal Coal Mine Health and Safety Act of 1969 (P.L. 91-173) established a coal mine dust standard (based upon the British data); mandated provisions for other safety and health standards; provided health surveillance, transfer rights, and rate retention for miners; provided federal compensation for "Black Lung"; and guaranteed NIOSH right of entry for further research in coal mining. In many ways, this Act served as a model for subsequent legislation and health standards from other exposures (see Table II-13).

Because of the importance of coal as an energy source, and because of our vast coal resources, coal dust and coal products (synfuels) will continue to be produced for decades to come (see Figure II-22 and Tables II-14 and II-15). Graphite and carbon black represent other important carbonaceous exposures found in dozens of commercial processes in several industries; these exposures will also continue and expand in the years to come. Therefore it is essential to understand the biological effects of these exposures and how these effects may be mitigated or prevented.

DEFINITION

In discussing coal workers' pneumoconiosis or pneumoconiosis arising from carbon dust exposure, it is essential to define pneumoconiosis and the popular term "Black Lung." Coal workers' pneumoconiosis (CWP) and carbon pneumoconiosis are specific diseases resulting from the inhalation and deposition in the lung of carbonaceous dust and the lung's reaction to the dust so deposited. In CWP, the disease is manifest characteristically by the coal macule and later by coal micronodules and nodules resulting in simple coal workers' pneumoconiosis. In some cases large (1-3 cm) lesions, or even massive consolidated lesions, develop resulting in progressive massive fibrosis (PMF).

"Black Lung" is a legislatively defined term which encompasses the classical medical definition of coal workers' pneumoconiosis, but is defined by the Act as "a chronic dust disease of the lung arising out of employment in an underground coal mine." (Title IV—Black Lung Benefits—Part A—Federal Mine Safety and Health Act of 1977, P.L. 91-173 as amended by P.L. 95-164). This definition is used to cover disability primarily from chronic airways obstruction which is associated with coal mine dust exposure. Tuberculosis *per se* appearing in a coal miner has not qualified for benefits, nor has the development of other bacterial or viral illnesses, or lung cancer. In practice, however, miners with these and other chronic lung conditions who meet any of the qualifying criteria in the Act—if in the judgment of the examining physician and administrative law judge they have developed their condition in association with coal mine employment—may be compensated for total disability. Medical costs for these conditions have been paid by the Department of Labor. Thus the definition of "Black Lung" is broad and imprecise: it will not be discussed further in this chapter.

Table II-13

COAL MINING HEALTH AND SAFETY
LEGISLATION IN THE UNITED STATES*

1865: Bill is introduced to create "Federal Mining Bureau." It is not passed.

1910: Bureau of Mines is established, but specifically denied right of inspection.

1941: Bureau of Mines is granted authority to inspect, but it is not given authority to establish or enforce safety codes (Title I Federal Coal Mine Safety Act).

1946: Federal Mine Safety Code for Bituminous Coal and Lignite Mines is issued by the Director, Bureau of Mines (agreement between Secretary of the Interior and the United Mine Workers of America) and included in the 1946 (Krug-Lewis) UMWA Wage Agreement.

1947: Congress requests coal mine operators and state agencies to report compliance with the Federal Mine Safety Code. Thirty-three percent compliance is reported.

1952: Title II of the Federal Coal Mine Safety Act is passed. All mines employing 15 or more persons underground must comply with the Act. Enforcement is limited to issuing orders of withdrawal for imminent danger or for failure to abate violations within a reasonable time.

1966: Amendments to 1952 law. Mines employing under 15 employees are included under 1952 Act; stronger regulatory powers are given to Bureau of Mines, such as the provision permitting the closing of a mine or section of a mine because of an unwarrantable failure to correct a dangerous condition.

1969: Federal Coal Mine Health and Safety Act is passed. In this Act the hazards of pneumoconiosis are, for the first time, given prominence, in addition to those of accidents.

1972: Black Lung Benefits Act of 1972 is passed. Several sections of Title IV are amended, liberalizing awarding of compensation benefits.

1977: Federal Mine Safety and Health Act of 1977 is passed. This Act amends Coal Mine Health and Safety Act of 1969 largely by adding health and safety standard setting, inspection, and research provisions for metal and nonmetal miners while leaving the 1969 Act largely intact. This Act also consolidated health and safety compliance activities for general industry (OSHA) and mining (MSHA) in the Department of Labor.

1977: Black Lung Benefits Revenue Act of 1977 was passed. This provided for an excise tax on the sale of coal by the producer to establish trust funds to pay black lung benefits.

1977: Black Lung Benefits Reform Act of 1977 was passed. This Act was passed to improve and further define provisions for awarding black lung benefits. Additionally, it established (a mandate) that a detailed study of occupational lung disease would be undertaken by the Department of Labor and NIOSH.

1981: Black Lung Benefits Revenue Act of 1981 was passed. This Act was passed to increase revenue for the Black Lung Disability Trust Fund, based on a new tax on coal with respect to sales after December 31, 1981.

*Source: (91)

Table II-14

U.S. COAL RESERVES

Coal Type	Short Tons
Bituminous	747,357 x 10^6
Sub-Bituminous	485,766 x 10^6
Lignite	478,134 x 10^6
Anthracite	19,662 x 10^6
Total	1,730,919,000,000

Source: (164)

Table II-15

U.S. COAL PRODUCTION FOR 1979 BY COAL RANK AND TYPE OF MINING

ANTHRACITE

Source	*Short Tons*
Strip Mine	2,962,000
Deep Mine	595,000
Culm Bank (Anthracite waste)	1,480,000
Total	5,037,000

BITUMINOUS, SUB-BITUMINOUS AND LIGNITE

Source	*Short Tons*
Strip Mine	462,324,000
Deep Mine	306,344,000
Total	768,668,000

*DOE Data

Source: (81)

Table II-16

POPULATION AT RISK TO EXPOSURE TO U.S. COALS BY PRINCIPAL WORK AREA

Work Area	Anthracite	Bituminous & Lignite
Underground Miners*	483	141,065
Surface Miners	1,625	69,214
Preparation Plant	1,116	22,235
Shop	80	2,729
Totals	3,304	235,243

*Includes Mine Construction

Source: Personal Communication HSCAC, MSHA, Denver Federal Center, February 19, 1981.

OCCUPATIONS AND INDUSTRIES INVOLVED

Within coal mining, exposure is commonly divided into underground and surface operations. Coal mine construction is included legislatively, and properly so, as an underground mining exposure (see Table II-16). Although most underground jobs result in heavier exposures to coal mine dust, certain surface jobs, particularly drillers, may have significant exposure to respirable coal dust and free silica.

Carbon black has been defined by the American Society for Testing and Materials as "a material consisting of elemental carbon in the form of near-spherical colloidal particles and coalesced particle aggregates of colloidal size, obtained from partial combustion or thermal decomposition of hydrocarbons." Carbon black is classified as furnace, thermal, or channel black depending on the manufacturing process (see Table II-17) (3).

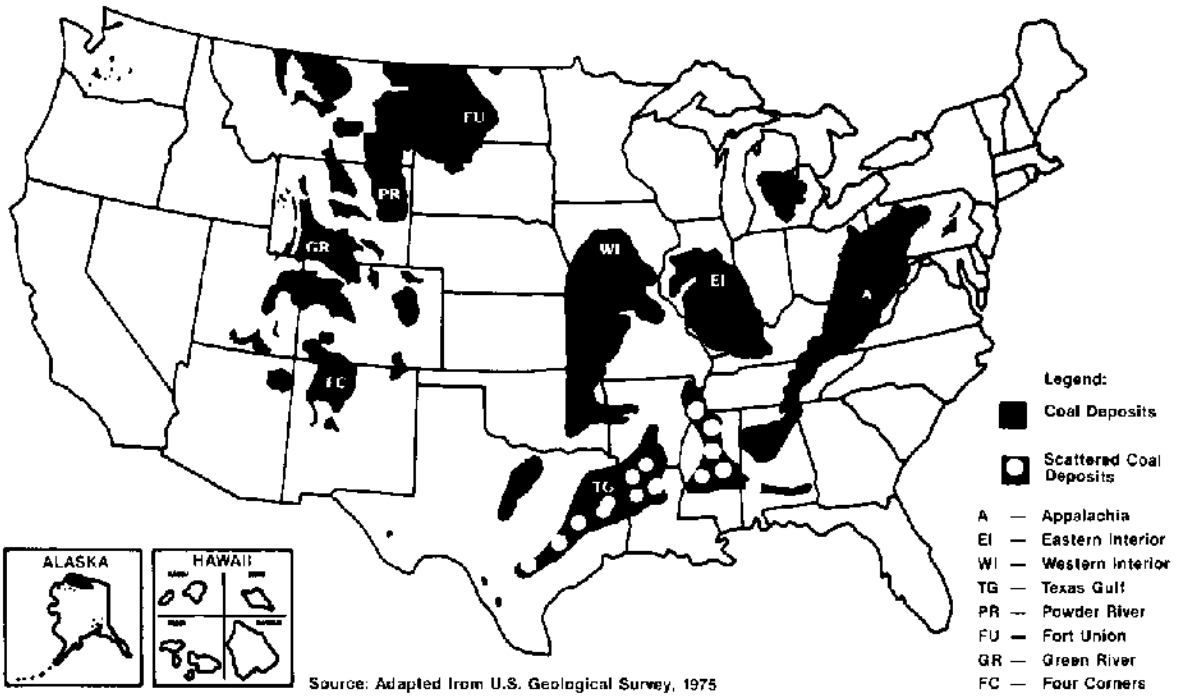
As of 1976 there were eight companies operating 32 carbon black plants in the United States. Of the 2,924 million pounds sold domestically, 2,720 million were used in pigmenting and reinforcing rubber, 80 million in inks, 19 million in paints, and 3 million in the manufacture of paper. The remaining 102 million pounds were used in plastics, ceramics, foods, chemicals, and other products. The worker may be exposed during any of a number of processes including production, pelletizing, screening, packaging, loading, and unloading (130).

Graphite is widely used in a number of industrial applications and is the third major form of carbonaceous dust exposure (see Table II-18). Graphite may be natural graphite, also called plumbago, or artificial or synthetic graphite. The difference is important from a respiratory disease standpoint in that natural graphite, which is mined from siliceous sediments (Sri Lanka, Madagascar, Italy, Brazil) may contain significant percentages of free silica (11% in Italian graphite) (138). Synthetic graphite contains only traces of free silica except for pyrolitic graphite which may contain significant amounts of quartz and cristobalite (137).

ESTIMATE OF POPULATION AT RISK AND DISEASE PREVALENCE

Because of the extensive regulatory and health surveillance systems mandated by the Federal Coal Mine Health and Safety Act of

Coal Deposits in the United States



Source: Adapted from U.S. Geological Survey, 1975

Figure II-22. Map of coal deposits.

1969, reasonably good estimates may be made in regard to underground coal miners. Table II-16 provides the most recent available figures on coal mining employment by type of coal mine and by type of coal. Estimates on the prevalence of CWP *per se* are provided from results of the third round of the NIOSH National Coal Workers' Health Surveillance Program (Table II-24). Similar estimates of CWP prevalence are not available for surface miners, as the Act did not mandate medical examinations for these miners and they are not yet covered by the health standard provision of the Act.

NIOSH estimates that approximately 2.4 million workers are potentially exposed to natural and synthetic graphites. Although pneumoconiosis, both simple CWP and PMF, is well documented in natural graphite workers and cited among synthetic graphite workers, the lack of epidemiological studies prevents making prevalence estimates.

NIOSH has estimated that 35,000 workers are exposed to carbon black directly or indirectly (130). There are only limited epidemiological studies available, making any estimate of carbon pneumoconiosis in these industries impossible.

EPIDEMIOLOGY

The epidemiological literature on coal mining is vast and represents, for the most part, the best epidemiology has to offer in defining occupational disease causality. Early observations were largely anecdotal but useful statistics were collected by about 1900. Since then, cohort mortality, cross-sectional, prospective and population based intervention studies have greatly expanded our understanding of the health effects of coal mine dust exposure. However, gaps remain in our understanding of certain aspects of coal dust induced diseases and in the quantitation and interaction of risk factors.

Historical Perspective

The three part series entitled "History of Lung Diseases of Coal Miners in Great Britain" published in 1951-52 by Andrew Meikeljohn provides an excellent review of the evolution of observations on coal workers' pneumoconiosis between 1800 and 1950 (109-111). Historical

Table II-17
PHYSICAL AND CHEMICAL PROPERTIES OF VARIOUS TYPES OF CARBON BLACK

Property	Furnace Black		Channel Black	Thermal Black
	Oil	Gas		
Composition				
Carbon (%)	98	99.2	88.4-95.2 (Avg. 91.2)	—
Oxygen (%)	0.8	0.4	3.6-11.2 (Avg. 7.8)	—
Hydrogen (%)	0.3	0.3	0.4-0.8 (Avg. 0.6)	—
Ash-Ca, Mg, Na (%)	0.1-1.0	0.1-1.0	0.01	—
Volatile matter (%)	1-2	1-2	5-18	1
Average particle diameter (μm) 0.14-0.47	0.018-0.06	0.04-0.08		0.01-0.03
Specific surface (Sq. mg/g)	25-200	25-50	100-1,000	7-13
pH	8-9	8-9	3-5	8-9
Benzene extractables (%) 0.03-1.75	0.05-0.1	0.05-0.15		—

Source: (130)

Table II-18
OCCUPATIONAL EXPOSURES TO NATURAL GRAPHITE

Refractory ceramics and crucibles
Foundry facings
Steel and cast iron manufacture
Pencils
Lubricants
Neutron moderators in atomic reactors
Electrodes
Electrotyping
Graphite mining and milling

Source: (137)

accounts often provide the most lucid descriptions of diseases and reveal insights into the pathogenesis and prevention of diseases that are often held to be recent advances. Such is the history of coal workers' pneumoconiosis.

Although coal was mined from outcroppings by the workers of Newbattle Abbey as early as 1291, significant occupational exposure to coal mine dust did not occur until after the invention of the steam engine which was first applied to underground mining in Scotland in 1762. By 1839, it was estimated that Britain was producing 36,000,000 tons of coal, and by 1866 over 100,000,000 tons were being mined by a work force of over 320,000 (109).

The first report of the Commissioners on Children's Employment in Mines (H.M.S.O., 1842) provides an early account of working conditions in coal mines. Employment was not confined to men but also included women and children. Ventilation was reportedly so poor and the air so thick with carbonic acid gas, gunpowder fumes, and smoke from oil lamps and tallow candles, that it was often inadequate to sustain hard labor or the lamps (109).

French pathological anatomists Bayle and Laennec described "melanosis" but did not associate these dark blue or black lungs with occupation (109). Pearson wrote the first English

Table II-19

OCCUPATIONS WITH POTENTIAL EXPOSURE TO CARBON BLACK

Battery workers
Carbon black workers
Carbon electrode workers
Carburization workers
Cement workers
Ceramics workers
Food processors
Ink workers
Paint manufacture workers
Paper workers
Plastic workers
Printers
Rubber workers

Source: (130)

paper on "melanosis" and linked it with the living environment (109). In one case, he further attributed this condition to tobacco smoking.

In 1831, Gregory published a "Case of Peculiar Black Infiltration of the Lungs Resembling Melanosis" based on his pathological observations on a 59-year-old collier (a coal miner) who died of progressive heart failure—the first case reported to arise from coal mine employment. He observed that this black infiltration differed from melanosis; was similar to a "form of phthisis" prevalent among "stone cutters, millers, and needle-grinders," and that it was possibly due to the inhalation of coal dust (109).

Among the clearest early descriptions of the pulmonary pathology found among colliers are those of Dr. William Craig of Glasgow. He clearly appreciated the importance of studying lungs fixed in inflation allowing assessment of dust deposition and emphysema (32):

> It is only in the case of colliers, molders, or others who make large quantities of black matter, that the lungs are reduced perfectly solid. The best manner of ascertaining the exact situation of the black matter in such cases is by inflating the lung slightly, drying it thoroughly, and then cutting it into slices in various directions. Where the lung is prepared in this manner, the air cells can be distinctly seen with the naked eye, and by means of a small magnifier the exact situation of the black matter may be easily ascertained.

The "Black Infiltration" was first described as "anthracosis" by Dr. Thomas Stratton in 1837, preceding by nearly thirty years Zenker's generic "pneumoconiosis" (159). Considerable clinical interest in this condition is evident from several accounts dating to this period. W. Thomson provides the following account of the clinical picture with advanced disease (161):

> He has a considerable degree of the stoop or rounded curvature of the back which is so frequently seen in old asthmatics, and his sternum and ribs are projected forwards in the manner in which they are usually seen to be in such individuals.

Pathological studies by Thomson and others described two types of "black infiltration" which closely approximate our current understanding of simple coal workers' pneumoconiosis and progressive massive fibrosis. Pneumoconiosis arising from working with stone as opposed to coal was recognized to be of different etiology and significance (109).

By 1851, the etiology and importance of respiratory disease among colliers was well enough established that physicians were calling for preventive measures in the mines. William Calder concluded his Edinburgh Clinical Club presentation with the following admonition (14):

> The disease, once established, does not admit of care or art. The only means of preventing the disease seems to consist of ventilating the mines where colliers work, or adapting a means of carrying off the fumes to which the moulders of iron and copper are exposed. Such prophylactic measures are equally called for, whatever theory of the nature of the disorder shall ultimately prove to be correct.

J. B. Thomson (1858) further observed (106):

> I cannot help thinking that medical inspectors should, long ere this, have been appointed to cooperate with mining engineers, in order to apply the most enlightened rules of hygiene for the safety and health of this numerous and important class of work people. Had this been the case, I am satisfied that the true cause or causes of their diseases would, ere now, have been much palliated or prevented.

In 1860 and 1861, Dr. Greenhow was assigned from the Privy Council to inquire into the excessive mortality among colliers from respiratory diseases (50). Greenhow, who made other important observations on bronchitis and respiratory disease among cotton textile workers, com-

mented on the "asthmatical symptoms" leading many to be "disabled at from 40 to 50 years of age." This is perhaps the earliest observation of the importance of airways disease among coal miners.

By 1871, there was clear evidence that steps were being taken to adequately ventilate the coal pits. Mr. Leonard Brough, H. M. Inspector of Mines for South Wales and Monmouth commented (12):

> Abundant ventilatory power, plenty of room for air in motion throughout the pit, attention to the state of the atmosphere, good officers and strict discipline; these are the arcana of safety underground, if indeed there are any secrets in the matter, which I very much doubt, for it is only a question of money after all.

Apart from ventilation, the importance of transferring affected miners out of dusty areas appears to have been appreciated as early as 1875. Therefore, the basic elements of a prevention program for respiratory disease among coal miners had been established more than a century ago.

In 1880, the first Workmen's Compensation Act was passed in Britain to provide compensation for accidents during employment. It was not, however, until 1904 that the first occupational disease, lead poisoning in pottery workers, was compensated (110). With better ventilation and shorter working hours, serious respiratory disease among coal miners appeared to decline markedly. This was supported by available Registrar-General data between 1871-1880. Based on this data and clinical observation, it was concluded that coal miner mortality only slightly exceeded that of a number of "healthy classes of men." Attention turned to silica exposure among cutlery grinders, earthenware workers, stone masons, and sandstone quarrymen with special attention to the importance of silica exposure and the risk from tuberculosis. Coal dust exposure, it was suggested, perhaps even retarded the development of tuberculosis (63). Still, it was appreciated that coal miners suffered more respiratory diseases than most other industries. Arlidge, in his text "The Diseases of Occupations," commented on the multiple causes for dyspnea among miners—deposition and "infarction" of the lung with "foreign matter," "bronchial trouble. . .with its attendant plugging with mucous and thickening of the lining membranes of the tubes," ". . .the greater or less extent of tissue in an emphysematous state," and "cardiac disease secondary to the respiratory disease" (4).

By 1906 a Departmental Committee on Compensation for Industrial Diseases was established to determine which diseases should be scheduled as occupational in nature, and despite conflicting testimony, found (110):

> We are clearly of the opinion that coal miners are not liable to fibroid phthisis, and although cases of anthracosis, using the term to mean cases in which the lung is charged with coal dust, are commonly met with, we cannot find that in any one that condition has proved to be a contributory cause of death.

This finding was reinforced by E. L. Collis in his 1915 Milroy Lectures on industrial pneumoconiosis (26):

> I have attempted to justify the claim that dust inhalation plays an important part in determining the occurrence of respiratory diseases. Some dusts such as coal, it is true, not only appear to have no power of producing pneumoconiosis, but even may possess some inhibitory influence on phthisis; but most dusts have an injurious influence, and of all dusts that of silica is most injurious.

However, thirteen years later Collis and Gilchrist published an important paper, "Effects of Dust upon Coal Trimmers," in which the mortality experience of Welsh coal trimmers was compared to all males in England and Wales (27). The low rate of "phthisis" was again noted, but only among younger miners—what today we would suspect to be a "healthy worker effect." Unusually high mortality was, however, observed for bronchitis and pneumonia. Further, chest radiographs, then only recently applied to the study of respiratory diseases, demonstrated radiographic changes previously widely regarded as only characteristic of silicosis. Because none of these men had previous occupational exposure to silica, this paper provided the basis for establishment of coal dust as a separate cause of pneumoconiosis. Also in 1928, coal miners were first included in the Various Industries (Silicosis) Scheme (established in 1919), but only coal miners thought to have also had silica rock dust exposure (11).

Among the many pursuits of the famous physiologist T. S. Haldane, was an interest in mining and respiratory diseases of miners. Haldane, in addition to his post as professor of physiology at Oxford, was honorary director of the Mining Research Laboratory in Birmingham. He

was largely responsible for the introduction of rock dusting as a means to prevent explosions. He also held strong views on the nature of respiratory disease among coal miners and strongly opposed the inclusion of coal miners' compensation for silicosis. He recognized that the radiographic appearance of "coal miners' pneumoconiosis" was indistinguishable from that of silicosis, but opposed making a diagnosis without benefit of occupational history. He was also convinced, prophetically it would appear today, that bronchitis among coal miners was a major source of their respiratory disease. In regard to bronchitis and dust exposure he stated that "it seemed practically certain that excessive inhalation of coal-dust or shale-dust must cause bronchitis and ought therefore to be avoided (111)."

The Various Industries (Silicosis) Scheme of 1928 was amended and extended in 1931 and again in 1934, to include all underground colliery workers. "Silicosis" claims among miners in South Wales increased markedly, yet many cases thought to be atypical were refused (111). As a result there was a public demand for scientific investigation which was begun in 1936 when the Medical Research Council was asked by the Home Office and the Mines Department to investigate chronic respiratory disease among coal miners, especially those in South Wales. Medical Research Council (MRC) Reports were issued in 1942, 1943, and 1945 (108). The findings reported by Hart and Aslett confirmed the importance of free silica exposure in the causation of classical silicosis, but found that the vast majority of pneumoconiosis cases arose from coal mine dust exposure—especially among those at the face, but also among others working underground, among those "screening" coal on the surface, and among coal trimmers. As a result of these studies, and the growing social and economic problems faced by coal miners, the Coal Mining Industry Pneumoconiosis (Compensation) Scheme was passed and became effective July 1, 1943 (111). In order to deal with questions raised about the treatment and rehabilitation of coal miners, the Minister of Fuel and Power appointed a committee which published a report one year later (114). Two important recommendations were to establish: 1) a rehabilitation and treatment (clinical) research center, and 2) facilities for pathological research into dust deposition and disease. The MRC Pneumoconiosis Research Unit was established under the direction of Dr. Charles M. Fletcher, in 1945.

Mortality Studies

The earliest reliable mortality data on coal miners date to the 1906 British Registrar General occupational mortality statistics for the years 1890-2 and 1900-2. These figures suggested that overall coal miner mortality was declining, a view consistent with the predominant clinical view that respiratory disease among miners was decreasing. However, in 1928, Collis and Gilchrist reported that coal trimmers, who were not exposed to the free silica encountered in mining, clearly had pneumoconiosis (27).

Cancer Mortality: Early mortality studies of miners concentrated on cancer mortality. This was the result of a follow-up to a study of cancer of the bladder and prostate in various occupational groups, by Kennaway and Kennaway. Their initial study focused on coal miner mortality from cancer of the larynx and lungs in England and Wales from 1921-32 (80). Low ratios of observed to expected deaths were observed for all airway categories. A later study by the same authors (79) suggested that miners with high rates of pneumoconiosis had low rates of lung cancer and vice versa. In order to test this reciprocal hypothesis, James (76) studied 1,827 coal miners with pneumoconiosis and 1,531 noncoal workers in South Wales. Miners with slight pneumoconiosis were found to have a lung cancer ratio similar to non-coal workers; however, with progressively more severe simple CWP and especially within PMF, cancer rates fell. This suggested to James that early death from pneumoconiosis was likely a factor in the reduction of cancer deaths among coal miners. Interest in the possible associations between coal mining and lung cancer was continued by the 1958 study of Doll, who also found reduced lung cancer mortality among coal miners in four South Wales Districts (35). Based upon Registrar General data, Goldman calculated SMR's for miners and ex-miners; again underground miners were found to have low lung cancer SMR's compared to SMR's for surface miners and for other cancers. Goldman, however, found that coal mining towns generally also had lower lung cancer rates (46). Liddell, then with the National Coal Board, also reported reduced SMR's for lung cancer, particularly for underground workers—it was, however, noted that

there were not reciprocal increases in neoplasms from other sites (94). Recently Jacobsen and Miller et al. have reported on large mortality studies of British miners and have found no evidence of an association between lung cancer and coal mine dust exposure, nor was there any evidence that miners with CWP were at increased risk (39) (115).

It wasn't until 1963 that similar mortality studies began in the United States. Contrary to earlier British observations, Enterline reported an excess of lung cancer for his coal miner cohort (SMR = 192) (38). In a later paper, in which data from the Society of Actuaries over the periods 1915-26 and 1927-35 were analyzed, Enterline reported an overall greater than twofold excess mortality for coal miners, but a slight decrease in cancer mortality (SMR – 80) (39). A later U.S. Public Health Service mortality study of 3,726 miners who had participated in the 1962-63 Public Health Service coal miner prevalence study, revealed a total of only 30 lung cancer deaths and an SMR of 67 (29). The authors concluded that Appalachian miners appeared to have lung cancer mortality rates similar to those in Great Britain.

The most extensive mortality study of U.S. coal miners was recently completed by Rockette (150). He studied a cohort of 22,998 miners who represented a 10% sample of members of the United Mine Workers of America Health and Retirement Fund. The cohort was defined as of January 1, 1959, and consisted of all those eligible for benefits in the sample. Major findings are presented in Table II-21. Although the overall SMR (101.6) did not deviate significantly from 100, it is somewhat greater than that expected in a healthy working population. As a group, chronic respiratory diseases (influenza, emphysema, asthma, and tuberculosis) were significantly increased. Although bronchitis was not increased, it was noted that all deaths attributed to chronic bronchitis also mentioned emphysema on the death certificate. Other non-malignant respiratory diseases were significantly increased and account for the excess SMR under "all other causes" in Table II-21. Accidents also resulted in a significant excess of deaths—most accounted for by mine accidents. *Ill-defined causes* were also significantly increased, a finding attributed by the author as possibly being due to the rural nature of the cohort. Major cardiovascular disease accounted for the largest proportion of deaths but resulted in fewer deaths than expected. Hypertensive heart disease and hypertension were the only categories with SMR's over 100; the latter was found to be significant when compared to 1965 U.S. white male mortality figures. The author urged caution in accepting this because of the racial composition of the cohort.

Malignant neoplasms accounted for the second greatest proportion of deaths, but the SMR for all malignant neoplasms did not deviate significantly from 100. Analysis by neoplasm site, relative to the 1965 total male population, revealed a modest increase in stomach cancer with a reciprocal decrease in colon cancer. Respiratory cancer, particularly lung cancer, was also slightly increased, while cancer of the genital organs was decreased. The modest, but significant increase in lung cancer was noted by the authors to be well within the variation in SMR expected by regional or smoking differences (smoking histories were not available) between the cohort and control groups.

Alternatively, the excess in stomach cancer is consistent with that of other investigators, most of whom have found moderate yet significant excesses (33)(75)(103)(158). Because stomach cancer may be related to diet, ethnic origin, and socioeconomic class as well as environmental factors, Matalo controlled for these factors in studying two Utah counties where coal is both mined and burned in the home and another Utah county where the population is similar but where coal was not mined or burned (103). Pronounced increases in stomach cancer mortality, especially among men, was observed in the coal county—a finding the authors concluded could in part be related to "coal carrying carcinogenic hydrocarbons." A subsequent study of gastric cancer mortality in 23 coal producing counties with control counties matched by socioeconomic indicators, also reported excess risk ratios for stomach cancer (33). These investigators, based on other cancer risk ratios which corresponded to patterns previously associated with socioeconomic class, concluded that the increase in gastric cancer was a likely reflection of socioeconomic class, even though the exposed and control groups were well matched for education and income. However, recent British evidence has suggested a dust relationship with gastrointestinal cancer and pneumoconiosis (75) (115).

Pneumoconiosis Mortality: Mortality studies have been more consistent and definitive in resolving other questions about coal miner mortality. Although most mortality studies have revealed increased SMR's for respiratory diseases and it was clear that much of this excess was due to pneumoconiosis, it was not clear as to whether simple pneumoconiosis *per se* resulted in increased mortality nor what other risk factors may have influenced mortality rates. Beginning with the studies of Carpenter et al. in 1956 (16), miners' mortality was studied in relation to the ILO radiographic classification of pneumoconiosis* (69). Carpenter found minimal increases in SMR when comparing Category O or Category 1, 2, 3, simple CWP, and Category A of PMF with non-miners. However, this was not true of more advanced PMF, which appeared to be associated with increased mortality. Cochrane, in 1964, and again in 1973 (20 year follow-up of miners of the Rhondda Fach, South Wales) confirmed that Category O miners had an SMR similar to those with Category 1, 2, 3, and A radiographs while those with Category B, C, and D had a marked increase in mortality (22). The mortality of Category O miners was, however, somewhat less favorable than for controls. Similar observations were made by Ortmeyer who studied both anthracite and bituminous miners (135). SMR's for simple CWP and Category A PMF were not increased among bituminous miners but were somewhat increased in anthracite miners. Other categories of PMF were clearly increased in SMR. However, a recent report from the Institute of Occupational Medicine in Edinburgh based on a National Coal Board cohort has now reported decreased survival for miners with Category 1 simple CWP (see Table II-20) (115). This is a new and—because of the size and length of follow-up of this cohort—important observation.

Other Observations: Ortmeyer also studied the effect of smoking and lung function on subsequent mortality in a 10 year follow-up of a cohort of 3,726 Appalachian coal miners (136). Both smoking and ex-smoking miners were found to have increased SMR's relative to nonsmokers and U.S. male controls. Miners with decreased lung function ($FEV_1/FVC \leq 70\%$) clearly had greater mortality, while those with lung function above this level appeared to have no increase in overall mortality. This finding has recently been confirmed by Miller et al. (115).

Costello, who studied the same cohort for cardiovascular disease, reported a deficit for cardiovascular disease among working miners and only a slight excess among nonworking miners (28). Obese miners who smoked were found to be a group with a particularly unfavorable mortality. Costello's results tend to agree with previous British studies and the findings of Rockette and Higgins but are inconsistent with the findings of Enterline.

Morbidity Studies

The modern era of investigations into coal workers' pneumoconiosis began with the observation of Collis and Gilchrist on the effects of dust on coal trimmers (27). This was a combined proportional mortality and clinical study of the Coal Trimmers' Union at Cardiff, South Wales. Review of death certificates revealed marked excesses of bronchitis and pneumonia, but unlike those with silicosis, no excess mortality from tuberculosis. Reviews of several case histories revealed a radiographic pattern like that of silicosis. From these observations arose the coal workers' pneumoconiosis disease entity as separate from silicosis.

Meanwhile public concern about respiratory disease among anthracite miners in Pennsylvania lead the Governor (in 1932) to request that the U.S. Public Health Service conduct a study into the "nature and prevalence of chronic incapacitating miners' asthma" (36)(165). A thorough study of 2,711 working miners and 135 disabled miners was completed in 1934 (see Table II-22). Over 23% of the working miners had radiographic evidence of "anthracosilicosis." A clear relationship between years underground and dust concentration was established and a dust standard of 50 million particles per cubic foot (mppcf) was recommended. Pulmonary infections were also observed to be related to length of service, as was the prevalence of tuberculosis (15% in early anthracosilicosis and 43% in more advanced states). Diagnostic criteria were established and severe cardiac disease (cor pulmonale) documented among the disabled miners. A series of recommendations were made and included dust control at the source, increased mine ventilation, environmental monitoring, TB testing and control, and periodic medical examinations with

*Since this is the most recent ILO Classification, this is the only one which will be referenced for this chapter. Readers are referred to the International Labour Office, Geneva, Switzerland, for earlier Classification schemes.

Table II-20

SUMMARY OF PREVIOUS MORTALITY STUDIES OF COAL MINER COHORTS*

Study	Method	Time	Study Group	Control Group	Comment
Kennaway and Kennaway 1936 (80)	Death certificates and census	1921-1932	Coal miners in England and Wales	General population in England and Wales	Lung cancer SMR = 59 Larynx cancer SMR = 53
Kennaway and Kennaway 1947 (79)	Death certificates and census	1933-1938	Coal miners in England and Wales	General population in England and Wales	Lung cancer SMR = 59 Larynx cancer SMR = 66
James 1955 (76)	Examination of the necropsies of miners with pneumoconiosis and of nonminers	1947-1952	1,827 miners with pneumoconiosis and 1,531 nonminers in South Wales	Relative risk based on the rate for all miners	Inverse relationship between the severity of pneumoconiosis and SMR for lung cancer. Very low lung cancer SMR with PMF suggests competitive risk
Carpenter et al. 1956 (16)	Cross-sectional study	1950-1953	Residents of the Rhondda Fach, South Wales	General population of England and Wales	MINERS AND EX-MINERS SMR Category 0 110.2 Category 1, 2, 3, A 111.0 Category B, C, D 138.6 Nonminers 104.1
Doll 1958 (35)	Death certificates	1948-1956	Coal miners in South	All workers in South Wales	Lung cancer SMR = 48; adjusted for other causes SMR = 58
Cochrane et al. 1964 (22)	6-year follow-up cohort study	1951-1956	Residents of the Rhondda Fach, South Wales	General population of England and Wales	MINERS AND EX-MINERS SMR Category 0 126.8 Category 1, 2, 3, A 119.2 Category B, C, D 209.2
Goldman 1965 (46)	6-year follow-up cohort study	1951-1956	Residents of the Rhondda Fach, South Wales	General population of England and Wales	Miners and Ex-miners lung cancer SMR = 81.1, nonminers lung cancer SMR = 53.1
Heasman et al. 1958 (55) Goldman 1965 (46)	Death certificates and information from the National Coal Board	1955	Coal miners of England and Wales	General population of England and Wales	SMR-Lung Cancer / Other Cancer Surface Workers 70.1 / 102.0 Underground Workers 91.5 / 113.4

*Source: (150) as modified

Table II-20

SUMMARY OF PREVIOUS MORTALITY STUDIES OF COAL MINER COHORTS (Continued)

Study	Method	Time	Study Group	Control Group	Comment
Stocks 1962 (158)	Death certificates and census	1949-1953	Coal miners and their wives in England and Wales	General population of England and Wales	SMR-Coal Miners / Their Wives Stomach Cancer 149 / 154 Bronchitis 135 / 175 Respiratory TB 119 / 145
Vinyard and Lieben 1960 (171) Lieben and Hill 1962 (92)	Death certificates	1957-1959 1959-1962	Deaths with mention of pneumoconiosis in Pennsylvania		Pneumoconiosis as primary cause of death: 63% Pneumoconiosis as contributory cause of death: 37% Among these: 49.1% with heart disease as primary cause 15.1% with cancer as primary cause 7.2% with pneumonia and influenza as primary cause
Enterline 1964 (38) PHS 1962	Death certificates and census	1950	Coal miners in United States	Males with working experience in U.S.	Cause of Death / SMR All Causes 195 Respiratory Disease 491 Stomach Cancer 275 Lung Cancer 192 CHD 144 TB 268 Accidents 286
Enterline 1972 (39)	Data from the Society of Actuaries	1949-1963	Policy holders in underground mining	Policy holders under standard risk	Cause of Death / SMR All Causes 172 Respiratory Disease 1,111 Cancer 80 Digestive Disease 260 Accident 626

Table II-20

SUMMARY OF PREVIOUS MORTALITY STUDIES OF COAL MINER COHORTS (Continued)

Study	Method	Time	Study Group	Control Group	Comment
Enterline 1972 (39)	28½ year follow-up cohort study	1937-1966	533 men working in coal mines in Beckley area in 1937	United States male population	Cause of Death: SMR All Causes: 157.9 Respiratory Disease: 150.0 TB: 173.9 Syphilis: 445.9 Lung Cancer: 110.9 Digestive Cancer: 210.0 Accidents: 269.0
Liddell 1972 (94)	Death certificates and information from the National Coal Board and a sample census of mining industry in 1961	1960	Coal miners in England and Wales	Occupied and retired males in England and Wales 1959-1963	Face Workers (SMR) / Other Underground Workers (SMR) / Surface Workers (SMR) Overall: 77 / 102 / 137 Lung Cancer: 49 / 53 / 82 Stomach Cancer: 101 / 128 / 32 Bronchitis: 26 / 64 / 129 Pneumonia: 25 / 58 / 132 Pneumoconiosis: 191 / 413 / 556 Accident (Excl. vehicle): 357 / 236 / 221
Matalo et al. 1972 (103)	Annual Gastric Cancer incidence based on Rocky Mountain States Cooperative Tumor Registry	1965-1969	Residents and coal miners of Carbon and Emery Counties	Residents of Utah Co. Utah, State of Utah, State of Connecticut	Threefold to fourfold increase in gastric cancer among residents, threefold to eightfold increase among miners.
Ortmeyer et al. 1973 (135)	A five year follow-up cohort study	1965/67-1970	Pennsylvania coal miners compensated for CWP	White males in Pennsylvania 1959-61	*SMR* / *Anthracite* / *Bituminous* Category 1,2,3,A: 108-119 / 82-105 Category B,C: 143-192 / 120-145

Table II-20
SUMMARY OF PREVIOUS MORTALITY STUDIES OF COAL MINER COHORTS (Continued)

Study	Method	Time	Study Group	Control Group	Comment
Creagan et al. 1974 (33)	Deaths obtained from National Center for Health Statistics. Population estimated from census	1950-1969	Residents in 23 coal mining counties in U.S.	Residents in non-coal mining counties in the same states and matched by educational level	Increase in risk ratio among coal county residents for lung and stomach cancer risk ratios, but decreased risk ratios for leukemia, colon, cervix, and breast.
Costello et al. 1974 (29)	10-year follow-up cohort study	1962/63-1971	3,726 Appalachian coal miners (the 1950 PHS CWP study)	Male population in United States 1968	Lung cancer SMR = 67; 22 of 24 lung cancer deaths had smoking history.
Ortmeyer et al. 1974 (136)	10-year follow-up cohort study	1962/63-1971	3,726 Appalachian coal miners (the 1950 PHS CWP study)	Male population in United States 1968	SMR for all causes = 104. Increased in association with smoking (current or ex-smoker), ex-mining status, and lung function ($FEV_1/FVC<70\%$).
Costello et al. 1975 (28)	10-year follow-up cohort study	1962/63-1971	3,726 Appalachian coal miners (the 1950 PHS CWP study)	Male population in United States 1968	*Cause of Death* / *Working Miners* (SMR) / *Ex-Miners* (SMR) All Heart Disease 73 104 Ischemic Heart Disease 73 105
Jacobsen 1976 (75)	Prospective cohort mortality study	1958-1972	Sample of National Coal Board cohort for whom vital status was known: a) 100% sample of those with CWP by radiograph (3,523) b) 50% sample of those remaining (13,786) Total cohort = 17,309	Registrar General mortality for all males—England Scotland, and Wales. Internal sub-group comparisons.	Cancer of digestive organs, respiratory disease and bronchitis mortality appear to be dose related. Simple CWP not associated with increase in mortality except among younger miners. Respiratory symptoms and smoking associated with increased mortality. No evidence of an association between lung cancer and coal mine dust exposure. (See discussion).

Table II-20

SUMMARY OF PREVIOUS MORTALITY STUDIES OF COAL MINER COHORTS (Continued)

Study	Method	Time	Study Group	Control Group	Comment
Rockette (150)	Retrospective cohort mortality	1959-1971	10% sample of 550,000 UMWA Welfare and Retirement Fund records yielding 23,233 eligible for benefits from the fund on January 1, 1959. Vital status determined as of December 31, 1971.	Total U.S. male population	Excess mortality from accidents; several categories of respiratory disease, and stomach cancer. (See Table III-21).
Miller, Jacobsen, and Steele (115) 1981	Prospective cohort study	1958-1980	Further follow-up of the Jacobsen cohort above 22 to 26 years from identification	As above	Miners with Category A PMF at the start of follow-up showed a considerably higher mortality than men without PMF. Survival rates for Category 1 simple CWP (all ages over 24) were reduced compared to men with Category 0 radiographs. There was no trend to increasing mortality with increasing category of simple CWP. Mortality from pneumoconiosis, bronchitis, and emphysema was more severe among those with heavy dust exposure before study start-up. Digestive system cancer was related to increasing dust exposure and increasing pneumoconiosis. No evidence of increased risk of lung cancer. Smoking miners had 5½ times the lung cancer mortality of nonminers (subgroup data). Those with low levels of FEV_1 had increased mortality attributable to lung cancer, bronchitis and emphysema (55 to 64 at start-up), pneumoconiosis, other respiratory disease, and ischemic heart disease.

Table II-21

OBSERVED AND EXPECTED DEATHS, AND STANDARDIZED MORTALITY RATIOS FOR COAL MINERS FOR SELECTED CAUSES OF DEATH (N = 22,998)

Cause of Death	Observed	Expected	SMR*
All Causes	7,628	7,506.1	101.6
All Malignant Neoplasms	1,223	1,252.2	97.7
Benign and Unspecified Neoplasms	14	14.4	97.5
Major Cardiovascular Diseases	4,285	4,501.2	95.2*
Bronchitis	27	31.5	84.8
Acute Bronchitis and Bronchiolitis	1	2.5	—
Chronic and Unqualified Bronchitis	26	29.0	89.7
Influenza	28	14.8	189.6*
Pneumonia	217	232.3	93.4
Emphysema	170	118.3	143.7*
Asthma	32	18.3	174.9*
Tuberculosis	63	43.3	145.5*
Syphilis	16	13.1	122.3
Other Infective and Parasitic Disease	13	17.6	74.1
Diabetes Mellitus	64	110.2	58.1*
Peptic Ulcer	42	58.7	71.6*
Cirrhosis of Liver	64	104.9	61.0*
Cholelithiasis, Cholecystitis, and Cholangitis	22	16.7	132.0
Nephritis and Nephrosis	42	46.2	91.0
Accidents	408	283.0	144.2*
Suicides	81	81.3	99.6
Homicides	30	26.1	115.1
Ill-Defined Causes	162	86.2	187.9*
All Other Causes	625	459.5	136.0*

*Standardized Mortality Ratio (SMR) is significantly different from 100 at the 5% level.
Source: (150)

an annual x-ray. Unfortunately, many of these recommendations were not adequately implemented and unlike Britain, where early studies led to industry-wide compensation programs and further epidemiologic investigation, neither broad legislation nor immediate further investigation followed. Indeed, studies by Clarke and Moffett in Southern Appalachia and the U.S. Public Health Service in Utah in the early 1940's, suggested that pneumoconiosis was relatively uncommon among bituminous miners (see Table II-22) (19)(42).

No other important epidemiological studies were published until 1963 when Hyatt studied a random sample of miners and ex-miners in Raleigh County, West Virginia (65). This study established, for bituminous miners of that region, the importance of coal mine dust exposure as a cause of both pneumoconiosis and lung impairment. Simple CWP was found in 46% and PMF in 7% of those surveyed and CWP was also found to be strongly related to years underground. Pulmonary impairment was observed among those with Category 3 simple CWP and PMF. Respiratory symptoms were found to be related to lung function which was itself related to years underground and unexplained by differences in age, smoking, or category of pneumoconiosis.

Thus, the severity of CWP and major risk factors among U.S. anthracite and bituminous miners were documented by 1963. The prevalence of the disease throughout the U.S. coal fields, quantitation of risk factors, and further evaluation of dose-response relationships awaited documentation by a flurry of epidemiological studies in Britain, Germany, and the United States.

Prevalence of CWP: A series of studies by the Pennsylvania Board of Health (93)(104)(105)

Table II-22

MORBIDITY STUDIES OF COAL MINERS IN THE UNITED STATES

Study	Date	Population Studied	Prevalence of CWP	Comment
Office of Industrial Hygiene and Sanitation U.S. Public Health Service Dreesen, et al. (36)	1934	Pennsylvania anthracite miners, 2,711 working in 3 representative mines; 135 disabled ex-miners	23.7% "anthrasilicosis" 100.0% "anthrasilicosis"	Documented the seriousness of CWP in United States. Established different dose-response relationships for coal and rock workers. Infection and cor pulmonale important complicating factors.
Clarke and Moffett (19)	1941	744 Southern coal miners of a single mine. Exposure 1 + years	3.1% "presilicotic" 1% "silicotic nodulation"	TB a common complication (7.4%). Clear increase in "nodulation" with years underground.
U.S. Public Health Service Flinn et al. (42)	1942	507 Utah bituminous coal miners	3.2% "anthracosilicosis"	13 of 16 cases with many years underground. No case with less than 10 years experience. Two cases with under 20 mppcf.
Hyatt, et al. (65)	1963	Stratified random sample of miners and ex-miners from Raleigh Co., W. Va. n = 267 ages 45-58	46% Simple and PMF 7% PMF	58% with respiratory symptoms associated with years underground but not CWP. Increased lung impairment with Category 3 and PMF. Decreasing lung function with years underground independent of smoking and aging.
Lieben et al. (93)	1961	4,182 Central Pennsylvania coal miners—medium and higher rank coal	30% overall 40% with CWP had PMF	Included working miners with 20 or more years of mining and retired miners. 25% participation.
McBride et al. (104)	1963	8,237 Western Pennsylvania bituminous coal miners—low rank coal	11% overall 37% with CWP had PMF	Included miners with 20 years or more mining and retired miners. 68% participation.

Table II-22

MORBIDITY STUDIES OF COAL MINERS IN THE UNITED STATES (Continued)

Study	Date	Population Studied	Prevalence of CWP	Comment
McBride et al. (105)	1966	1,858 anthracite miners participating in Pennsylvania Division of Occupational Health Surveys	30% overall 10% < 30 years work 50% > 40 years work 80% > 40 years work and retired	A third of the men without CWP reported severe dyspnea. Cough related to years exposure and age. 70% of men over 55 had FEV_1 < 70%.
U.S. Public Health Service Lainhart et al. (87)	1962-65	Sample of 97 bituminous Appalachian coal counties, large and small mines: working miners = 617 nonworking miners unemployed = 617 pensioned = 574. Pulmonary function on: working miners = 2,342 nonworking miners = 1,028	Working Miners / Non-Working Miners No CWP 84.9 / 74.9 Suspect 5.3 / 6.9 Simple 6.8 / 9.2 PMF 3.0 / 9.0	Association between dyspnea and productive cough and years underground and smoking. Years underground appeared to influence lung function more than smoking. Moderate effect of bronchitis and CWP on lung function. Marked association between dyspnea and lung function.
U.S. Public Health Service Enterline (40)	1963-65	Community study of Mullens and Richwood, W. Va., with a matched sample of miners and ex-miners and nonminers and their wives from each community	Mullens = 13.85% Richwood = 6.3%	Excessive respiratory symptoms among miners and wives relative to nonminers and nonminer wives. Reduced lung function among miners only in Mullens not Richwood. No difference in lung function of wives.
Higgins, et al. (61)	1968	Cross-sectional study of three mining communities in Marion Co., W. Va. n = 957	Simple CWP = 29/448 PMF = 4/448	Except among older miners, respiratory symptoms and FEV_1 did not differ between miners and nonminers. Lung function in this West Virginia sample appeared to be better than two British communities studied earlier.

Table II-22

MORBIDITY STUDIES OF COAL MINERS IN THE UNITED STATES (Continued)

Study	Date	Population Studied	Prevalence of CWP	Comment
U.S. Public Health Service First Round National Study of Coal Workers' Pneumoconiosis (131)	1969-71	29 bituminous and 2 anthracite mines selection based on seam, mining method, number of miners, and retrospective dust data. n = 9,076	Total PMF Anthracite 60.0 14.3 Appalachia 30.0 2.1 Midwest 25.0 2.9 West 10.5 0	Respiratory symptoms and lung function associated with region, years underground, and smoking.
U.S. Public Health Service Second Round National Study of Coal Workers' Pneumoconiosis (132)	1972-75	Same mines as in round 1. Some mines closed and Some new mines selected n = 9,343	Category 0 92.0 1 4.9 2 1.9 3 0.2 PMF 1.0	Previous findings confirmed. Flow rates, especially of larger lung volumes, associated with years underground and bronchitis and smoking. Time between rounds precludes meaningful assessment of progression.
Higgins, et al. (59)	1963-72	Follow-up community of Mullens and Richwood. Miners and ex-miners and nonminers.	*Mullens* *Richwood* Miners 18.1(4.5) 9.9(1.5) Ex-Miners & Non-Miners 3.8(0.3) 0.3(0.0)	Significant increase in persistent cough, phlegm, and wheeze associated with mining and smoking. Clear effect of mining on decreased lung functions. Mining effect over 9 years as great or greater than smoking effect on decline in FEV_1.

documented an increasing gradient in CWP prevalence from western to eastern Pennsylvania —from 11% in the West to 35% in Central and Eastern Pennsylvania mines. The U.S. Public Health Service studied 97 Appalachian coal counties and found nearly 10% of working miners and 18% (9% with PMF) of nonworking miners to have CWP (87).

The NIOSH National Study of Coal Workers' Pneumoconiosis was the first U.S. industry-wide study of this disease (131). It was designed as a prospective study of some 30 mines and over 9,000 miners and included PA and lateral chest radiographs, spirometry, and a standard MRC questionnaire. CWP prevalence in the first round of this study, which ran from 1969 to 1971, anteceded outward migration of a substantial proportion of miners with many years underground and relatively high dust exposures. Still, the prevalence of CWP reported was a good deal higher than previously reported (see Table II-22). This may in part be attributable to the use of new international standards (UICC 1968) for the interpretation of radiographs for the pneumoconioses and reader variation. In the second round of this prospective study, the study population was quite different due to the outward migration of older miners and an influx of young miners (7)(113). International radiographic standards for the interpretation of the pneumoconioses were again changed (1971 ILO/UC Classification), as were the radiograph readers. The change in radiographic standards is thought not to have contributed to lower prevalence rates (132). Evidence is presented elsewhere from the National Coal Miner Health Surveillance Program, that the prevalence of CWP by years underground did not differ between round one and round two; thus, reduction in dust level during this brief interval did not appear to influence CWP prevalence (113). Recent surveillance results are presented in Table II-24 and suggest a slight decrease in CWP prevalence—below 5% among working miners. Whether this is in part due to dust controls implemented in the early 1970's, or principally attributable to shifts in the mining work force, is not yet clear.

Dose-response studies: Dreessen and colleagues in their 1934 study of anthracite miners not only documented a marked associated between prevalence of "anthracosilicosis" and years underground, but also documented a clear dust and pneumoconiosis relationship (165). Men exposed to between 5 and 99 million dust particles per cubic foot of air over 25 or more years underground were found to have an approximate 7% prevalence of pneumoconiosis. This prevalence increased in each dust category to reach nearly a 90% prevalence over 25 years among men exposed to 300+ million dust particles per cubic foot of air.

Numerous later cross-sectional studies confirmed this observation using years underground as a surrogate for dose (see Table II-22). It was, however, not until the first 10 years of observations were completed by the Pneumoconiosis Field Research (PFR) Unit in Great Britain, that dose-response based on gravimetric sampling of respirable dust was established (73). That study was based on findings among 4,122 coalface workers at 20 collieries of the original 25 selected in 1953. Results of that study suggested a negligible risk of developing Category 2/1 CWP over a working lifetime below 2.0 mg/M^3 (See Figure II-23). These results were provided to the U.S. Congress and were instrumental in the mandating of the 2 mg/M^3 standard by the 1969 Act (see Table II-13). Jacobsen et al., also reported that smoking did not significantly affect the attack rate of pneumoconiosis or significantly alter the dose-response relationship (71).

The 20 year follow-up of miners in 10 of the PFR original collieries was recently reported (64). The original dose-response relationship was confirmed unambiguously, but the long-term risks were slightly greater (one to two percentage probability units) than estimated in 1969 (see Figure II-23). Large variations were noted between collieries and were not accounted for by quartz content, coal rank, or any other risk factors measured. Further, there was no pattern to suggest that quartz affected the probability of developing simple CWP. Jacobsen (1980) has recently, however, reported an association between PMF and quartz content based upon a case-control study arising from prospective data (72). In 1982, results from the third round of the NIOSH National Worker Health Surveillance Program and National Study of CWP, together with extensive dust data available from MSHA and NIOSH, should provide a U.S. assessment of approximately 10 years of exposure (following the 1969 Act).

Table II-23

RESPIRABLE DUST LEVELS (Mg/M^3)
HIGH RISK AND SELECTED OCCUPATIONS

	1968	1973	1976	1977
Jacksletter		4.2	1.9	1.9
Longwall		2.6	1.8	2.2
Continuous Miner Operator	6.5	2.1	1.3	1.3
Roofbolter	3.9	2.1	1.4	1.2
Cutter Operator	5.9	1.8	1.2	1.1
Loader Operator	6.0	2.7	1.2	1.3

Source: (113)

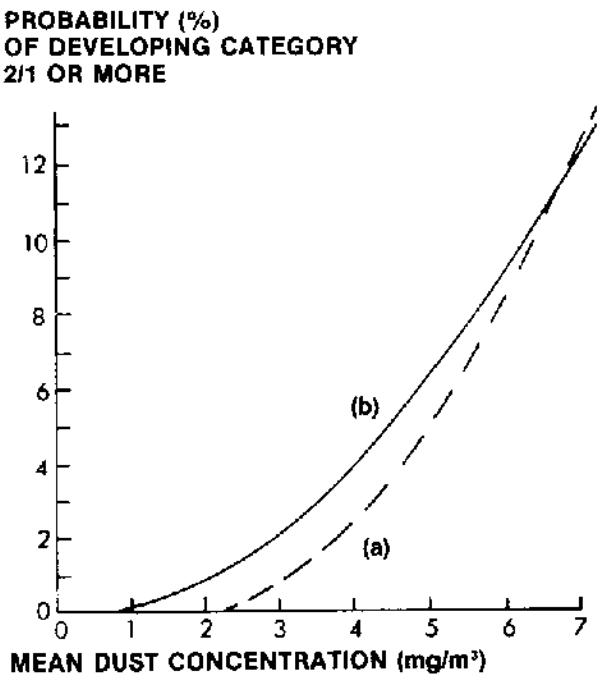


Fig. II-23. Lines (a) and (b) are estimates of probabilities of developing Category 2 or 3 of simple pneumoconiosis over an approximately 35-year working life at the coalface, in relation to the mean dust concentration experienced during that period. (a) is based on 10 years of data, Interim Standards Study, Pneumoconiosis Field Research. (b) is update of (a), based on 20 years of data, Pneumoconiosis Field Research.

Bronchitis: Historically, bronchitis and associated respiratory infections have been prominent respiratory findings among miners exposed at high dust levels. This has, however, not been a uniform finding in retrospective cohort mortality studies and has not been confirmed (nor looked at closely) in pathological studies. Respiratory infections and bronchitis were prominent findings in the 1934 U.S. Public Health Service study of anthracite miners (165). The symptoms of chronic cough, phlegm, and dyspnea have been consistently increased among underground miners and have usually been associated with years underground and smoking in more recent cross-sectional studies (40)(65)(87)(131)(132). Hyatt observed that many miners appeared to acquire their symptoms within the first year of employment and retain them for years following (65). Ashford et al. (1970) made similar observations on approximately 30,000 miners studied by the Pneumoconiosis Field Research Unit of the National Coal Board (6). They suggested that respiratory symptoms (persistent cough, persistent phlegm, breathlessness, and wheeze) were associated with radiographic evidence of pneumoconiosis, smoking, and age (neither years underground nor dust levels were controlled). They further suggested that those with bronchitis (persistent cough and phlegm) differed from those with breathlessness and wheeze. Rae et al. assessed chronic bronchitis and dust exposure among the 4,122 coal-face workers previously studied by Jacobsen, thus providing the first good estimate of dust exposure for such an assessment (142). A statistically significant association was found between increasing respirable dust concentration and increasing bronchitis among men in the 25-34 and 35-44 year age groups. Smoking and the presence of pneumoconiosis was associated with increased bronchitis prevalence in all age groups. The association between smoking and phlegm production, and the

Table II-24

COAL WORKERS' PNEUMOCONIOSIS IN ROUND THREE OF THE NIOSH NATIONAL COAL WORKERS' HEALTH SURVEILLANCE PROGRAM*

Years Mining	CAT 0	CAT 1	CAT 2	CAT 3	CAT PMF	Total
0-9	39,050 98.90%	416 1.05%	14 0.04%	1 0.00%	2 0.00%	39,483
10-19	6,167 93.185%	424 6.40%	22 0.332%	1 0.015%	4 0.060%	6,618
20-29	2,228 82.396%	410 15.163%	52 1.923%	5 0.185%	9 0.333%	2,704
30-39	2,094 71.737%	628 21.514%	130 4.454%	9 0.308%	58 1.987%	2,919
40+	369 66.606%	135 24.368%	32 5.776%	4 0.722%	14 2.527%	554
Total	49,908 95.467%	2013 3.851%	250 0.478%	20 0.038%	87 0.166%	52,278

*10/1/78 through 12/15/80

attack rate of CWP has also been studied by Jacobsen and colleagues (71).

Higgins conducted four mining community studies of respiratory disease in Britain (62) and the United States (60)(61). Excesses in bronchitis and breathlessness among miners and ex-miners were common findings, although the excesses were small in the U.S. study (Marion Co., W. Va.). Kibelstis and colleagues, based on questionnaire data from the first round of the NIOSH National Study of Coal Workers' Pneumoconiosis, reported a clear trend in bronchitis prevalence (83). Hankinson et al., in an analysis of second round data from the National Study of Coal Workers' Pneumoconiosis, demonstrated a bronchitis effect on lung function, particularly flow rates at higher lung volumes, in addition to an independent smoking effect (51). Rogan et al. reported similar effects. Rogan suggested that symptoms, once contracted, caused further reductions in lung function, even after dust exposure ceased (152). Finally, in a follow-up study of an earlier U.S. Public Health Service study in Mullens and Richwood, West Virginia, Higgins reported increased respiratory symptoms among miners and ex-miners as compared to nonminers (59). He concluded that independent and additive smoking and mining effects were operative.

Exposure to diesel emissions, a potentially important hazard, is now being observed with greater frequency in U.S. underground coal mines. Although American mines have traditionally been electrically powered, foreign mines have utilized diesel haulage equipment for decades. Possible diesel related health effects among coal miners have only recently been studied. Reger et al. studied over 800 underground U.S. coal miners whom he matched to other miners with similar smoking and dust exposures but who had worked only in electrically powered mines (147). Diesel exposed miners were found to have more bronchitis, which increased with years underground, and lower lung function than the reference group. However, surface miners at diesel mines also showed similar, but smaller trends, making interpretation difficult. The authors suggested that caution should be used in introducing diesels pending prospective epidemiological evaluation and other studies.

Lung Function: Seriously impaired lung function was recognized among miners with advanced CWP and especially PMF at the time

of the initial Public Health Service studies in 1934 (165). It was, however, not until the 1950's that pulmonary function testing in epidemiological surveys of working populations began contributing important quantifiable data on lung function. Hyatt and colleagues were the first to apply sophisticated lung function measurements in an epidemiological study of American coal miners (65). A volume displacement body plethysmograph was used and FEV_1, FVC, MMEF and RV/TLC ratio were reported. A marked association was found between all symptoms measured (phlegm, cough, wheeze, aggravation of symptoms by weather, episodes of cough and phlegm, and dyspnea) and all four measures of lung function. Increasing numbers of symptoms were similarly associated with a progressive decline in all four measures of lung function. Years worked underground was found to exert a significant and independent deleterious effect on lung function. Smoking was found to also be an important risk factor which appeared to work in an additive fashion to increase respiratory symptoms and decrease lung function.

These general observations have since been confirmed by larger, more representative cross-sectional and prospective studies of coal miners and studies of coal mining communities. Studies of lung function included in the Public Health Service study of 97 coal-producing Appalachian counties confirmed the findings of Hyatt in regard to the influence of years underground on FEV_1 and FVC and the additive effect of smoking (87). It was concluded that smoking did not exert the major effect in this study. A review by Higgins (1971) of chronic respiratory disease in four mining communities—while controlling for other important lung function risk factors—concluded that excesses in respiratory symptoms and decreased lung function (FEV_1) were commonly found among miners and ex-miners compared with nonminers living in those communities (60).

Ventilatory capacity and lung volumes were studied in the first round of the National Study of Coal Workers' Pneumoconiosis by Morgan et al. (122). No relationship between ventilatory capacity and radiographic category of simple CWP was found; however, PMF was associated with definite impairment. In addition, significant geographic variation (lung function tending to be worse among anthracite miners and best among western miners) was observed.

In an effort to better characterize the nature of lung function changes and the contribution of bronchitis among working coal miners, Hankinson et al. drew four well-matched groups of 428 miners from a total of over 9,000 working miners studied in the second round of the NIOSH National Study of Coal Workers' Pneumoconiosis (51). This provided a bronchitis and smoking specific analysis in which age and height were matched and allowed comparison and quantification of FEV_1, FVC, peak flow, four measures of flow rates ($\dot{V}max$ 25, 50, 75, 90), RV, and TLC. While both bronchitis alone and smoking alone produced significant adverse changes in most measures of lung function, smokers with bronchitis consistently showed the most impairment among these four groups. Smokers had greater decreases in flow rates at higher lung volumes and greater increases in RV and TLC, suggesting a prominent small airway or alveolar process, while measures more indicative of large airway function (FEV_1 and $\dot{V}max_{25 \text{ and } 50}$) were relatively more marked among those with bronchitis.

Quantitation of dust and smoking effects on declines in lung function over time have been attempted, based on the British Pneumoconiosis Field Research (PFR) data. An initial report by Rogan and colleagues was based on a sample of face workers who had been followed for at least 10 years by the PFR (152). Based on these results it was calculated that the effect of smoking 15 cigarettes/day over 40 years appeared comparable to experiencing a dust concentration of 14 mg/M^3 over that same period. The authors cautioned that this estimate may have underestimated the dust effect because of the highly selected nature of the sample. One criticism of the statistical analysis of that paper was the method used to standardize for smoking, even though there was no evidence of a significant interaction on lung function between smoking and dust exposure (21). Reanalysis of this data by smoking groups confirmed that pooling smoking and nonsmoking miners tended to underestimate the smoking effect with increasing age (75). Cochrane further suggested that a follow-up study of ex-miners in various regions would provide a better estimate of risk factor effects (21). Such a follow-up study was completed by the Institute of Occupational Medicine and reported in 1979. This sample was based on 12 of the original 24 mines studied, and of 3,870 miners examined, 2,094 were ex-miners

at the time of follow-up (112). Analysis of this data found that the effect of dust exposure on FEV_1 was at least half as much again and perhaps as much as twice as marked as the original Rogan estimate. Thus, the concern expressed by Rogan in regard to the selected nature of the face-worker sample was well justified.

The relative importance of coal mine dust exposure and smoking on decline in lung function among coal miners has been an important but controversial issue. It was reported, and is often quoted, that the effect of smoking is five times that of coal dust (83). Morgan asserts in a recent review (117) that "smoking has about five to ten times greater effect on ventilatory capacity," and more recently that "cigarette smoking is between six and ten times more important," citing the aforementioned review (83) (118). Yet examination of the original Kibelstis et al. data does not provide unqualified support for that conclusion. An assumption was apparently made that surface miners have negligible coal mine dust exposure. Such an assumption is faulty on two counts; one, because many surface miners have had significant previous underground exposure, and two, because certain surface jobs themselves result in significant dust exposure. Thus, there is not an adequate unexposed control.

However, if one takes the same data from the first round of the National Study of Coal Workers' Pneumoconiosis and compares it to the NIOSH external blue collar control population* (using the same methods as used in the NSCWP), and applies the spirometric standards for significant impairment used by the Department of Labor (FEV_1 ≤60% predicted), one finds an estimated relative risk for smoking of 2.0 and for dust exposure, 2.1; i.e., their effects on this indicator of impairment appear to be similar. It is important to note that the first round of the NSCWP was conducted prior to general availability of federal benefits which provided the means for many impaired miners to leave mining.

Higgins et al. provide prospective data, which is externally controlled, in their study of Mullins and Richwood (59). In this study, separate mining and smoking effects were documented. The annual decline in FEV_1 was slightly greater for smoking (smoking nonmining men compared to nonsmoking nonmining men) than the annual decline in FEV_1 for all miners compared to all nonminers.

These observations are consistent with the prospective British studies. As previously reviewed, reanalysis of the Rogan et al. data (152) suggested that the smoking effect had been underestimated (75). However, analysis of a less selected cohort, which included ex-miners, found that the effect of coal mine dust exposure was one and one half to two times as great, but that the reduction in FEV_1 attributable to smoking was no more marked than found previously by Jacobsen (70)(112). From review of these studies it appears that the smoking to dust effect ratio decline in FEV_1 is between one and two. It must be stressed that these are average values based on population studies. Older miners who have many years underground may have significantly more dust exposure than the average and therefore may be expected to be more affected by dust than smoking. Alternatively, younger miners will likely experience much less dust exposure; hence without similar reductions in smoking exposure, smoking would be expected to become a much more powerful risk factor. It should further be noted that one should not be surprised that a very high proportion of impaired coal miners have a history of cigarette smoking (112). This follows from two well established findings: 1) that over 80% of U.S. coal miners have a smoking history, and 2) that smoking significantly adds to the effect of dust exposure on decline in lung function.

In summary, coal mine dust exposure has been found to have an unambiguous, adverse effect on lung function, which is separate and additive to that of the other major (controllable) risk factor, smoking. Quantitation of the relative contributions of cigarette smoking and coal mine dust exposure have been difficult because good dust data is usually not available and because study populations are invariably selected (and thus effects underestimated) to some extent. It is safe, however, to conclude that both coal mine dust and cigarette smoking are important risk factors which operate additively to decrease lung function in a dose-response fashion.

Epidemiology of Graphite and Carbon Black Exposure: While both graphite pneumoconiosis and carbon pneumoconiosis are well documented clinically, there is only limited epidemiological data. It is known that graphite miners may develop pneumoconiosis, both simple and PMF, after 15 years exposure. Most of these reports have come from Sri Lanka (Ceylon)

*Technical Report to Director, NIOSH: Epidemiologic Study of Normal Blue Collar Workers, September 1981.

which is a major exporter of natural graphite (34)(144). Ronashinha and Uragoda studied a large graphite mine employing 344 underground and surface workers (144). Pneumoconiosis, which was described as being composed of both rounded and irregular opacities in mid and upper lung zones, was reported in 22.7% of those surveyed. Digital clubbing was found in 21.9% and specific attention was drawn to the absence of bronchitis and tuberculosis. Tuberculosis was reported among three miners in another mine but was not considered to be an increased hazard in graphite mining because of the relatively low silica content (163).

Among the reported respiratory effects of carbon black exposure are pneumoconiosis, bronchitis, and emphysema (130). Most of the epidemiological studies have involved limited numbers of workers. Dust exposures have been relatively high, or not reported, making interpretation difficult (130). Retrospective cohort mortality studies have raised the possibility that carbon black may itself be a carcinogen or bind other carcinogens (66-68). Apart from the clear evidence that carbon pneumoconiosis may occur among those exposed to carbon black, there is little morbidity data available. Valic reported a radiographic study of 35 Yugoslavian workers exposed to carbon black an average of 12.9 years (respirable dust concentrations = 7.2 ± 1.8 mg/M^3) (169). A risk-factor-matched, non-exposed control group was also studied. Initial lung function testing (FEV_1 and FVC) on both groups in 1964 revealed no significant differences. However, when tested again in 1971, a significant decline in lung function was noted in those exposed compared to controls. Although the authors raised questions about the adequacy of the control group (they lived near the carbon black plant), the declines in lung function among carbon black workers were still significantly greater than the decline expected in normal populations over time. Valic also reported that 17.1% of the 35 workers showed evidence of a fine nodular pneumoconiosis in the mid and lower lung zones.

PATHOLOGY AND PATHOGENESIS OF COAL WORKERS' PNEUMOCONIOSIS

The primary lesion of coal workers' pneumoconiosis (CWP) is the coal macule. This lesion was first clearly described by Gough (47) and by Heppleston (58). Before this time, opinions on specific lung pathology in coal workers were confused. Recognition and clear description of the pathology depended on the development of methods for examining sections of whole lung fixed in inflation (49). Using this technique, this earlier work has been confirmed in all the major coal producing nations. In whole lung sections, the coal macules appear as black areas 1-4 mm in diameter (Figure II-24). The smaller ones are usually circular while the larger ones are more irregular and often stellate. The lesions are usually symmetrically distributed in both lungs with a greater concentration in the upper lobes. Adjacent to the macule the airspaces are enlarged, constituting focal emphysema. These two features were considered pathognomic for coal workers' pneumoconiosis by the Pneumoconiosis Committee of the College of American Pathologists (85). Focal emphysema usually only involves a region of 1-2 mm around the pigmented macule. Both the macule and the associated focal emphysema occur in the region of the first order respiratory bronchioles. Microscopically, the pigment is found within macrophages both inside the airspaces and in the connective tissue around the respiratory bronchioles (Figure II-25). In longitudinal sections the pigment-laden cells thinly sheathe the walls of the bronchiole and may be relatively inconspicuous. In transverse section, the lesions are much more obvious. The macrophages are often densely packed and completely fill the airspaces. A fine reticulin fibrosis is present in the lesion. Collagen is either absent or sparse. There is loss of elastic fibers in the airways involved in the emphysema. In coal workers who have lived in retirement for many years, the alveoli within a macular lesion may no longer be totally filled with pigment-laden macrophages suggesting some degree of clearance (Figure II-26). The macules are nonetheless recognizable by the pigment in the interstitial tissue and the surrounding focal emphysema. A minor vascular change associated with the coal macule has been reported (128). This consists of an increase in arterial medial muscle due to hypertrophy of the muscle fibers as the artery transverses the lesion. The authors did not consider these vascular changes to have much functional significance. The mechanism whereby the coal macule develops is considered to be an overwhelming of normal lung particle clearance mechanisms by the heavy dust burden experienced. The reticulin fibrosis and possibly the emphysema may be due to the damaging effects of released macrophage

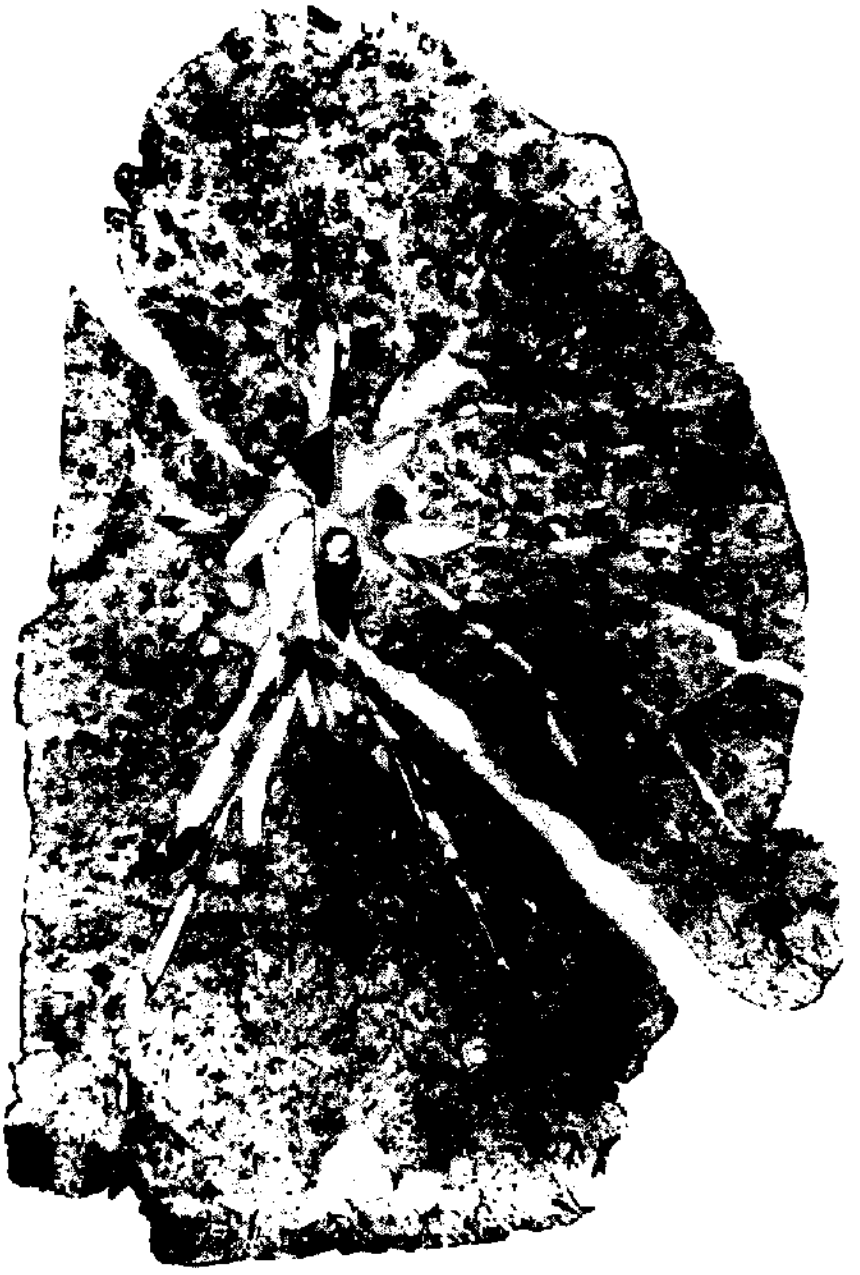


Figure II-24. 57-year-old coal miner who worked 31 years underground as a trackman, smoked 20 cigarettes per day. Whole lung section shows mild simple coal workers' pneumoconiosis. The macules, which are more numerous in the upper zone, are outlined by mild focal emphysema.

Figure II-25. High power micrograph of alveolar macrophage within alveolus from the lungs of an active miner. The majority of the phagocytosed particles are coal mine dust. Hematoxylin and eosin × 585.

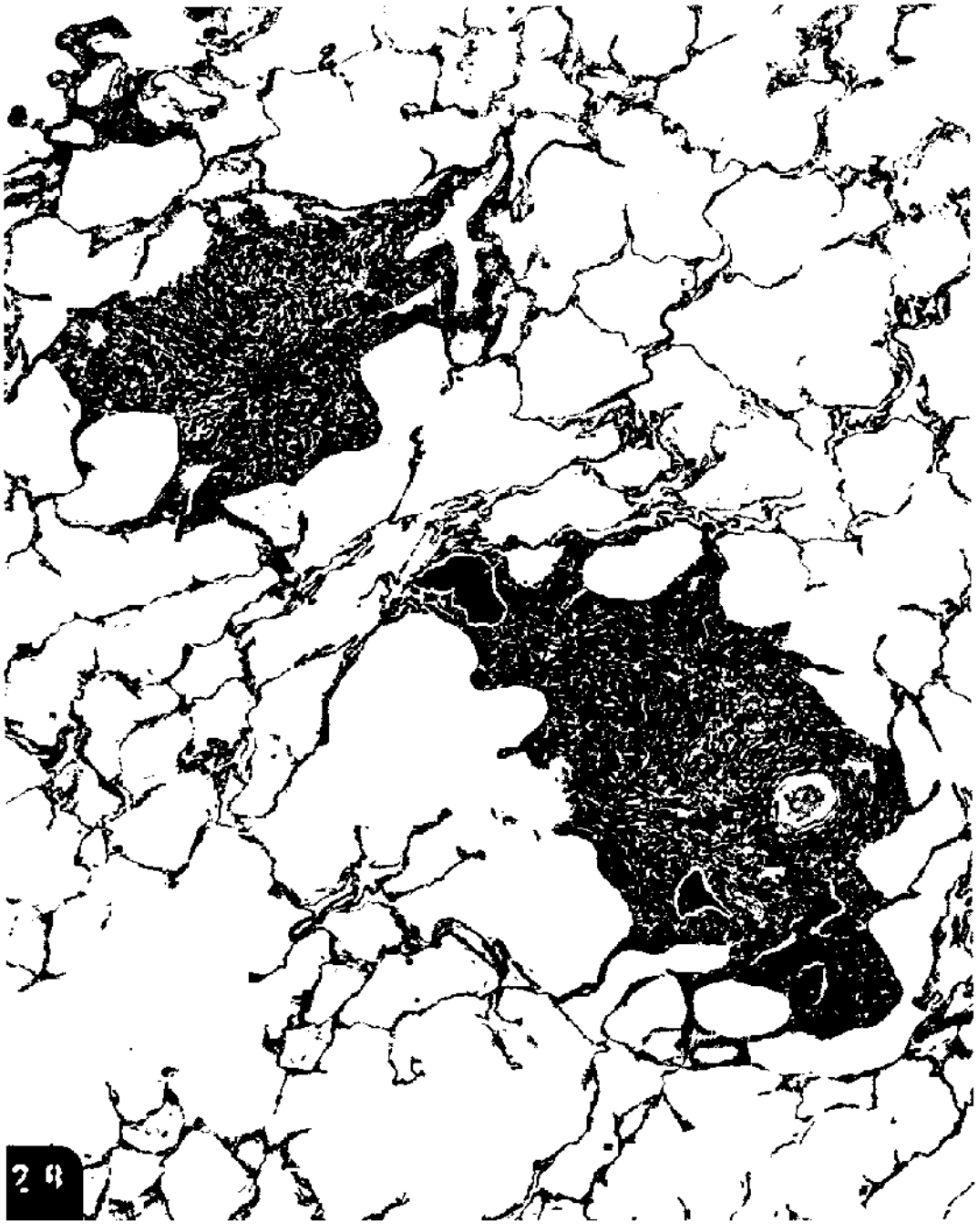


Figure II-26. Coal macules in the walls of respiratory bronchioles. The macules are composed of macrophages, coal mine dust and reticulin. There is minimal air space enlargement (focal emphysema) around the macule. Hematoxylin and eosin × 100.

lysosomal enzymes.

Even in nonoccupationally dust exposed individuals, pigmented lesions are found in the lung. This is presumed to be derived from atmospheric pollution and is more marked in urban as compared with rural dwellers. These "normal" pigment lesions vary in size up to about 4 mm. The pigment is contained within macrophages both on the walls of alveolae and in the stroma of the bronchiolar wall. In differentiating these lesions from the typical macule of coal workers' pneumoconiosis, three factors are useful. The "normal" pigment lesion is not usually associated with focal emphysema and the alveolae are not completely filled with pigment-laden macrophages. In addition, the pigment particles in the nonoccupationally exposed individuals are black and rounded while coal dust particles are angular, often translucent, and of a lighter yellow or brown color.

The coal macule is soft and is not palpable on examination of unfixed lung. This differentiates coal macules from the nodular lesions of CWP. A number of different nodular lesions are recognized in the lungs of coal workers. These are micro and macronodular lesions of simple coal workers' pneumoconiosis, progressive massive fibrosis, Caplan's lesion, and because miners are also exposed to crystalline silica from the strata surrounding the coal seam, the nodules of simple and conglomerate silicosis. In addition, the nodular lesions of the infective granulomata (tuberculosis and histoplasmosis, in particular) occur in coal miners, as in any other population, and require differentiation by classical histologic and microbiologic methods.

The nodular lesions of simple CWP are palpable because of their collagen content and sometimes because of calcification. They are divided for descriptive purposes into micro-nodular (up to 7 mm in diameter) and macro-nodular (greater than 7 mm in diameter) (85). The nodules are dark gray or black and are usually centriacinar in location (Figures II-27A and II-27B). Less frequently nodules are in the subpleural or peribronchial regions. They are often rounded but may have irregular prolongations into the surrounding tissue. They are commonly, but not invariably, associated with scar emphysema which in some individuals can be extensive. On microscopy the major difference between the nodular and macular lesions is the larger size and presence of hyalinized collagen in the former (Figure II-28). The collagen bundles are usually arranged in an interlacing or haphazard pattern and this feature is useful for distinguishing the nodules from silicosis where the collagen is concentrically arranged. The nodular lesions are believed to develop from macules but it is clearly impossible to demonstrate such progression in an individual coal worker. The reasons why nodules contain more collagen are not known, but the possibility that silicates—or the more toxic silica—are involved must be considered.

Some occupations within the mine are more commonly exposed to silica. These are the roof-bolters who are exposed to dust derived from the overburden, the motormen who, in some mines, used sand to provide traction for their vehicles, and drillers in surface coal operations (the latter may have acute silicosis). Such coal workers may have the classical lesions of silicosis. These nodules have smoother borders than the typical nodular lesions of CWP and, though they may contain black pigment, are often paler and may have a relatively pigment-free center (Figure II-29). They are more difficult to cut than CWP lesions. The arrangement of collagen is orderly and has a laminated or whorled pattern. Coalescence of adjacent silicotic nodules may occur resulting in conglomerate silicosis. Calcification in the hilar lymph nodes is common in silicosis. It may be of the egg shell type as described on radiologic examinations or may be randomly distributed.

Complicated coal workers' pneumoconiosis or progressive massive fibrosis (PMF) is diagnosed radiologically when an opacity 1 cm or greater is found. This conforms to the anatomic definition set out in the regulations of the National Coal Workers' Autopsy Study (166). However, the Pneumoconiosis Committee of the American College of Pathologists recommended a 2 cm standard as being more appropriate for pathological studies (85); a 3 cm standard is used in England (77). PMF occurs on a background of simple CWP, more commonly of the nodular rather than the macular type. The extent of simple CWP is usually considerable, but can consist of relatively few nodules and macules in some individuals. The lesions of PMF are most commonly situated in either upper lobe or the apical segments of the lower lobes. The lesions are usually asymmetrical in the two lungs.

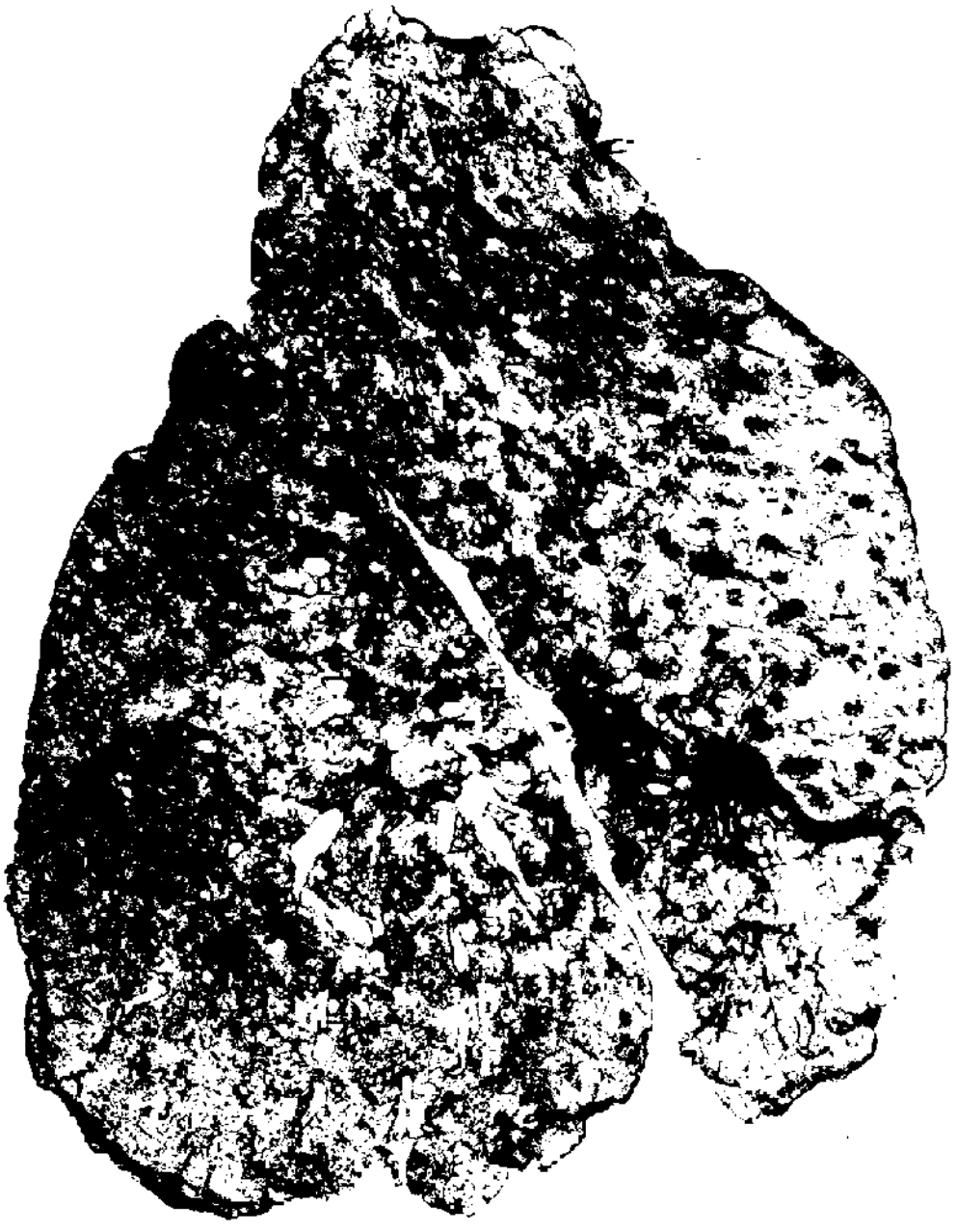


Figure II-27A. Coal miner, no detailed history available. (A) Whole lung section shows macules, micro and macronodules, confluent nodules, and a small PMF lesion. Mild focal, scar and paraseptal emphysema is present.

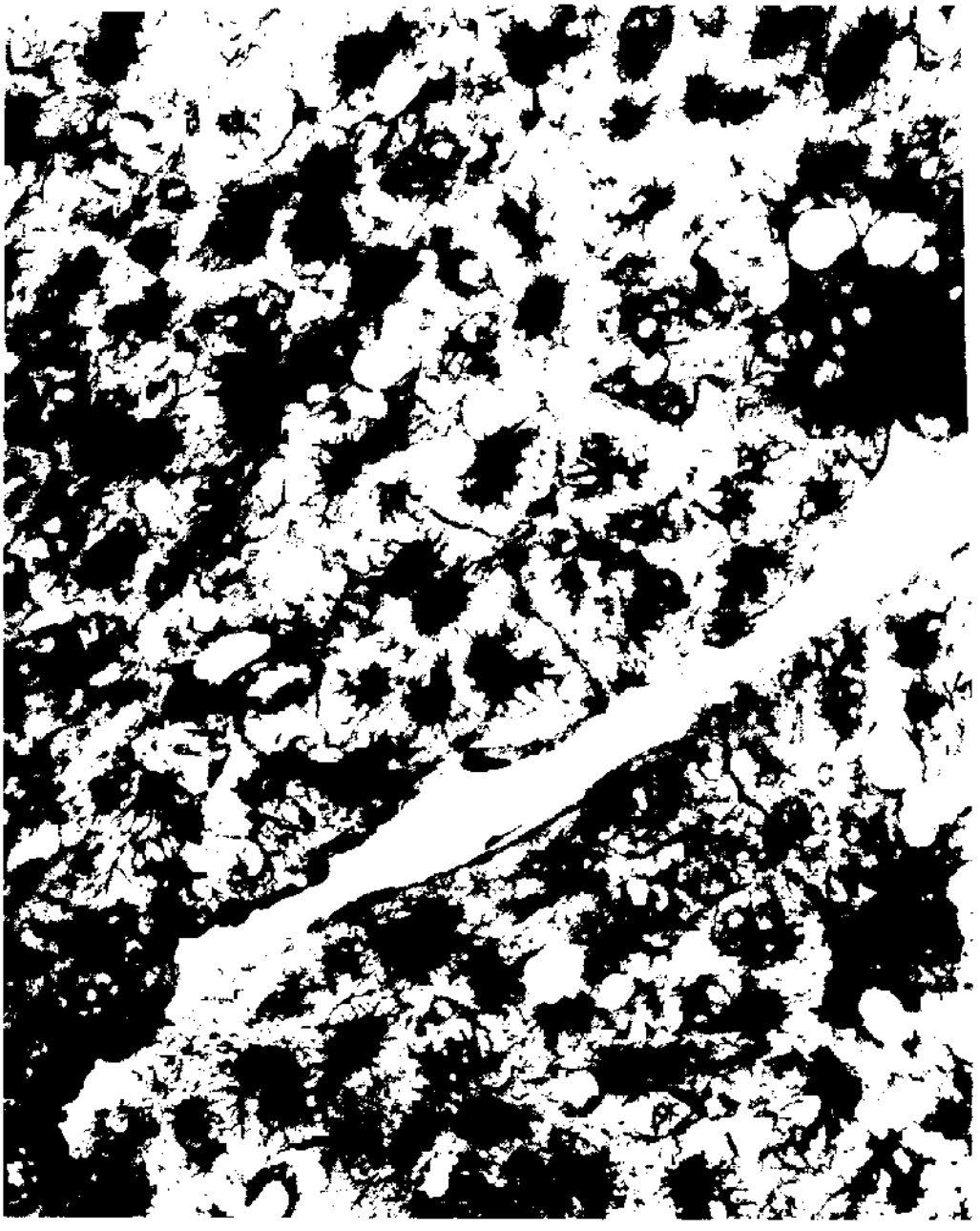


Figure II-27B. Close-up of micronodules and macules.

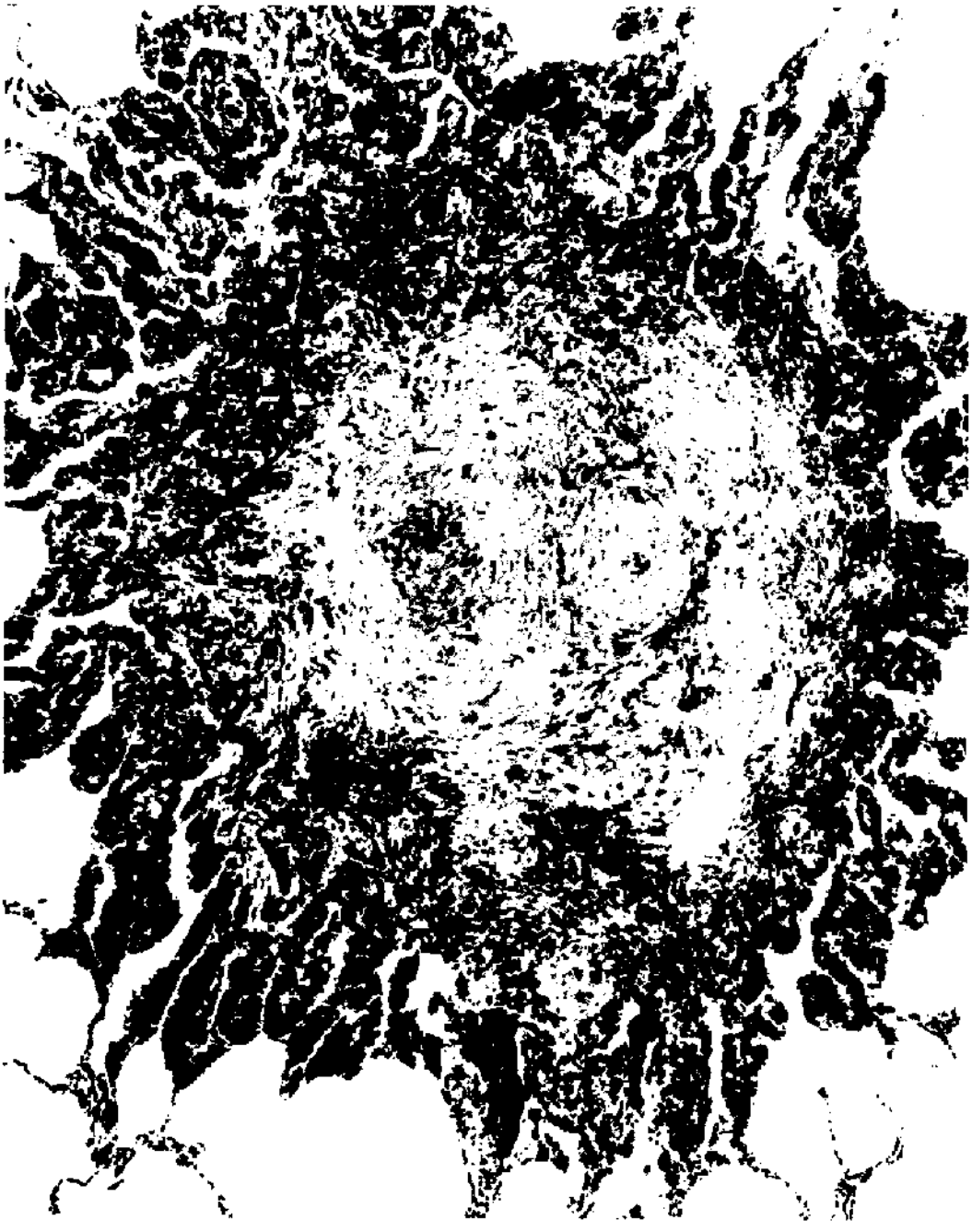


Figure II-28. Micronodule, composed of macrophages, dust and collagen. Hematoxylin and eosin × 250.

Figure II-29. Silicotic nodule in the lungs of a coal worker. The nodule has a hyalinized center with concentrically arranged collagen fibers. The majority of the coal dust is at the periphery of the lesion. Hematoxylin and eosin × 150.

They may cross interlobar fissures and often involve the pleura, when firm adhesions to the chest wall develop. Despite the name PMF, the lesions may or may not progress once exposure to dust has ceased, and certainly with a minimum size of 1 cm, are not necessarily massive. Truly massive lesions which replace more than a whole lobe do, however, occur. The cut surface of a PMF lesion presents a homogeneous black appearance. The texture of PMF is rubbery rather than gritty or hard when cut. The margins of the lesions may be smooth or have fibrous prolongations into the surrounding tissue. Emphysematous change is common in the lung around a PMF lesion, but occasional lungs show remarkably little emphysema even in the presence of large PMF lesions. The center of the lesion of PMF may have cavities containing opaque black liquid. Histologically, the periphery is composed of irregular reticulin and collagen interspersed with black pigment (Figure II-30). Moving away from the edge, recognizable collagen becomes more scanty and amorphous material containing coal dust is seen. This material has been found to contain principally an insoluble protein of unknown origin, calcium phosphate, and coal dust (173). Blood vessels and airways transversing the lesion are destroyed.

There have been several hypotheses which attempt to explain the mechanisms involved in the generation of progressive massive fibrosis. These have involved the role of tuberculosis, of silica, of immunologic factors, and of genetic predispositions. None of these hypothesis is entirely satisfactory and the mechanisms involved are still not fully understood.

Studies of coal miners, particularly in South Wales, revealed a strong association between tuberculosis and progressive massive fibrosis. Often the infection could only be diagnosed by examination of tissue at autopsy and in life the prevalence of sputum samples positive for *Mycobacterium tuberculosis* was much lower. Thus Rivers et al. found only 1.1% of positive sputa in miners with CWP as opposed to an isolation rate of 29% from autopsy material taken from 153 miners with coal workers' pneumoconiosis (149). The isolation rate for miners with PMF was 35% as opposed to 11% of those with simple CWP.

In experimental studies, Zaidi et al. showed that administration of coal mine dust and tubercle bacilli of low virulence resulted in massive fibrosis of the lungs in guinea pigs while neither alone produced this effect (176). This observation and the demonstrated association of tuberculous infection and PMF led to the hypothesis that PMF resulted from modification of the tissue response to coal dust by the infection. Several studies indicate that the development of PMF from simple CWP is not due to tuberculous infection. In a long-term study it was shown that decreasing the incidence of tuberculosis in a mining community did not result in a decrease in the attack rate of PMF (20)(24). Anti-mycobacterial chemotherapy was shown not to influence the course of early complicated CWP and some lesions progressed when not exposed to coal dust and while receiving chemotherapy (8). Thus, though associated in the past, tuberculosis does not have a clear etiologic relationship with PMF.

Because silica is a well-recognized fibrogenic agent, some researchers have suggested that silica in coal mine dust is responsible for the development of PMF. Evidence to support this hypothesis is limited and the occurrence of PMF in carbon electrode workers (174) and in coal trimmers who are exposed only to coal dust rather than coal mine dust (47) would support the view that silica is not necessarily involved. Comparison of the silica content of the lesions of PMF with that found in the rest of the lung did not show significant differences (129). Pratt criticized this conclusion because the silica concentration was related to tissue dry weight (141). He explained that when using this method of expressing results, the dry weight of tissue would increase because of tissue changes and, therefore, the concentration of silica expressed in milligrams silica per gram tissue would give a falsely low expression of true silica content. He was able to recalculate the data of Nagelschmidt et al. and by expressing the results as silica per whole lung, assuming that the lung was either wholly PMF or wholly non-PMF, showed that the silica content of PMF tissue was significantly higher than "the rest." The difference was of the order of twice. While this reopens the question of the role of silica in PMF, it certainly does not answer it.

Experimental animal studies have not been very helpful. Administration of coal dust with varying concentrations of silica to rats has shown little fibrosis with up to 10% of added silica

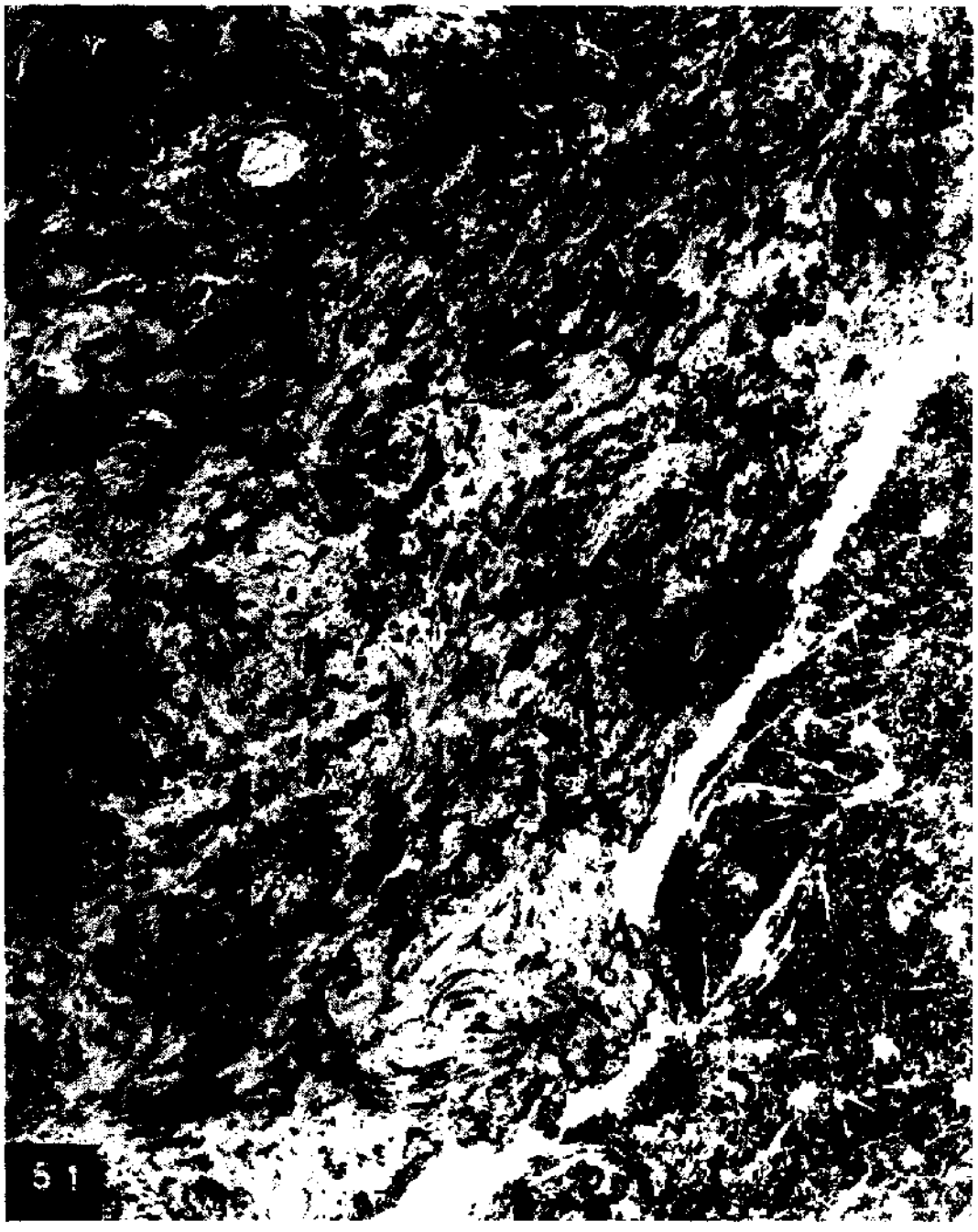


Figure II-30. Section from center of PMF lesion showing masses of black pigment embedded in bundles of haphazardly arranged collagen fibers. A cavity containing free dust and cholesterol crystals is seen at bottom right. Hematoxylin and eosin × 150.

(153). Martin et al. carried out studies for a longer period of time and showed that after 18 months exposure, 5% quartz plus coal dust was about three times more active in generating fibrosis than coal dust alone (102). Intratracheal injection of coal mine dust obtained from the lungs of deceased miners containing 0.7% and 1.6% quartz also produced significantly more fibrosis after 18 months than demineralized dust (84). These studies show that the silica in coal mine dust is fibrogenic but its fibrogenic potential is mitigated (suppressed) by other minerals within the coal mine dust. The relationship of these experimental studies to pathogenesis of PMF is, however, obscure as the lesions induced more closely resemble the nodular lesions of simple CWP rather than those of PMF. Thus they support the role of silica in the generation of nodular lesions of CWP but do not help in understanding the mechanism involved in the development of PMF.

The association between rheumatoid arthritis and complicated pneumoconiosis was first noted by Caplan (15). The lesions—Caplan's lesions—could usually be differentiated from typical PMF on radiologic examination and were histologically different. However, because of this observation, the possible role of immunologic phenomena in the generation of PMF attracted some research. Miners with PMF were found to have a higher prevalence of circulating rheumatoid factor than those with simple pneumoconiosis. Rheumatoid factor was found in plasma cells present in the walls of blood vessels in relation to the PMF lesions (172). This was not an all or nothing situation: they found tissue rheumatoid factor in 20% of simple pneumoconiosis lesions as opposed to 67% of PMF lesions with histologic vasculitis. Rheumatoid factor was found in only 19% of lungs with PMF without histologic vasculitis, suggesting perhaps there were two variants of PMF, one being an atypical Caplan's syndrome. The association of circulating rheumatoid factor with complicated pneumoconiosis was not confirmed in a study carried out in Pennsylvania and West Virginia miners although a high prevalence of antinuclear antibody was observed (95). Antibodies reactive with insoluble lung antigens have been suggested as possible modifiers of the tissue response to coal dust (13). Such antibodies, possibly reactive with either collagen or reticulin, have been demonstrated in the sera of a limited number of miners; however, there is no clear evidence on which to base an etiologic rôle in PMF.

The possibility that genetically determined mechanisms of tissue response to coal dust were involved in complicated CWP was investigated by comparing the frequencies of HLA histocompatability antigen in miners with simple, complicated and with no pneumoconiosis (56). An association between HLA-1 and resistance to the development of both simple and complicated CWP was suggested. An association between resistance to the development of PMF and antigen W18 was also suggested but further work has not supported this observation (57).

The mechanism whereby progressive massive fibrosis develops from simple pneumoconiosis is thus still not known. It is possible that PMF is not a single entity and that multiple mechanisms are involved.

As already noted, Caplan's lesions is another possible diagnosis for a nodular radiological opacity in a coal miner's chest x-ray. The lesions are described as well-defined, rounded nodular masses 0.3 cm to 5 cm in diameter and usually situated more peripherally than other nodular lesions of CWP. Most commonly described in European miners, they appear to be relatively rare in the United States (10). Histologic description is provided by the studies of Gough et al. (48). Caplan's lesions are round or oval and may contain cavities. They characteristically show a concentric arrangement of lighter and darker layers. The pale areas may contain clefts and calcification and are more common than in other lesions of CWP. Pallisading fibro-blasts, characteristic of many rheumatoid lesions, may be seen but are not prominent. There is characteristically a peripheral zone of active inflammation and vasculitis of local blood vessels. These histological features do not involve all pneumoconiotic lesions in a single lung and typical non-Caplan's lesions may be present side-by-side with typical Caplan's lesions. This indicates that local as well as systemic factors play a part in the generation of the lesion.

Emphysema, i.e., the enlargement of airspaces distal to the terminal bronchiole, is a common finding in the lungs of deceased coal miners. In any nonminer population studied, emphysema of all types will also be found. A classification of emphysema is provided by the re-

Figure II-31. 74-year-old coal miner who worked 27 years underground as a loader, smoked 20 cigarettes a day for 40 years. Whole lung section shows an area of PMF in the upper lobe set against a background of macular and nodular lesions of simple CWP. The lung also shows moderately severe emphysema and enlarged, deeply pigmented, peribronchial lymph nodes.

port of CIBA guest symposium (18). Only two types of emphysema can be definitely (pathologically) related to the inhalation of coal dust. These are the focal emphysema associated with the coal macule and scar emphysema related to the nodular lesions (Figure II-31). Focal emphysema usually involves only a small portion of the proximal part of the acinus and its limited extent suggests that it has little functional significance. More extensive, destructive, centriacinar emphysema is seen in a certain proportion of miners' lungs (154). These miners are usually cigarette smokers and it is difficult in an individual case to determine the relative importance of coal dust and cigarette smoke in the pathogenesis of these lesions. However, a recent study of coal miners with pneumoconiosis suggests that centrilobular emphysema may play a more important role in coal miners' lung impairment than previously appreciated (100). Although the numbers were viewed as too small to draw definite conclusions, smoking and nonsmoking miners were found to have similar degrees of centrilobular emphysema; before death, smokers were found to have lower lung function which the authors suggested was probably attributable to airway changes. The extent of the scar emphysema associated with nodules and PMF is highly variable. This suggests either other extraneous factors or individual variations in response are involved in its generation.

Chronic bronchitis, characterized by hypertrophy and hyperplasia of the bronchial mucous glands and goblet cell metaplasia of the small airways, is found in some coal miners' lungs. In general populations, these changes are associated with cigarette smoking. There are currently inadequate pathological data to relate the histologic changes of chronic bronchitis and coal dust inhalation; the entity "industrial bronchitis" is defined only by epidemiologic studies.

CLINICAL DESCRIPTION

Signs and Symptoms

The signs and symptoms of coal workers' pneumoconiosis (CWP) are the same as those which may occur with common nonoccupational lung diseases and are not, therefore, pathognomonic. One must rely on epidemiological studies to help determine to what extent clinical findings and pulmonary function test abnormalities can be attributed to coal mine dust exposure, and in an individual case (particularly if a smoker) this determination can be difficult. A detailed history and physical, looking for other treatable conditions such as asthma or congestive heart failure, are mandatory.

If one accepts a strict radiologic or pathologic definition of CWP in the absence of other lung disease, most authorities would agree that simple CWP *per se* causes few, if any, signs or symptoms (124)(137). However, there is evidence that coal mining exposure is a risk factor in chronic bronchitis and emphysema. Chronic bronchitis is defined as persistent cough and phlegm production, and emphysema, if marked, is associated with significant airways obstruction, dyspnea, and disability.

While results of some research questioned that causal relationship between coal mining and chronic bronchitis, more recent work has validated this association (see Epidemiology). Nonsmoking miners may show an increased prevalence of cough and phlegm production, but it is not known whether this industrial bronchitis can produce airways obstruction of a degree sufficient to cause dyspnea. Indeed, there is some evidence to suggest that even in smokers, chronic mucus hypersecretion *per se* plays little role in the development of chronic airflow obstruction (41). In working miners, studies by Hankinson et al. show small, but statistically significant spirometric differences between bronchitic and nonbronchitic groups, similar in magnitude to the smoking effect at larger lung volumes (52)(53).

It is controversial whether simple CWP or coal mining exposure causes disabling emphysema. The main studies supporting this concept come from one British group (96-98)(154). From their pathological studies the authors conclude that in their study populations, CWP usually caused progressive impairment of ventilation and that in these cases the presence of emphysema was found to be a more important determinant of this impairment than the radiologic category of simple CWP. The later study found substantial ventilatory impairment in a group of legally disabled miners including those with simple CWP. Several criticisms have been made of these papers, including a possible selection bias by studying miners who had been certified as disabled, inclusion of cases with progressive massive fibrosis (PMF), and a lack of correlation between pulmonary function decrement and x-ray category (45)(123)(134). A recent paper

by Lyons and colleagues has again reported that centrilobular emphysema is as common among nonsmoking miners as smoking miners (100). The authors added that the number of cases was small, making definite interpretation of the role of emphysema in CWP and among coal miners still uncertain.

There is, however, general agreement that complicated CWP (PMF) frequently produces substantial symptomatology and impairment, often associated with emphysema. Particularly in Categories B and C of PMF, cough, sputum production, and some degree of dyspnea are common. Melanoptysis, the often dramatic production of several ounces of black inky sputum from a ruptured lesion, can be considered the only specific, although somewhat unusual, clinical sign of CWP. On physical exam one may find evidence of collapse and consolidation as well as the decreased breath sounds, prolonged expiration, and adventitial sounds found in obstructive airways disease. In severe cases, signs of pulmonary hypertension, right ventricular hypertrophy, and congestive failure (cor pulmonale) may be present. PMF may also produce signs of restrictive pulmonary disease.

For proper medical management as well as correct disability evaluation, it is important to know which signs and symptoms should not be attributed to CWP or coal mining exposure. Thus chills, fever, night sweats, anorexia, weight loss, and usually finger clubbing require other explanations. Chest pain or hemoptysis, particularly in simple CWP, should prompt further diagnostic evaluation, just as it would in a nonminer.

Natural History

Coal workers' pneumoconiosis takes years to develop. In one large British study of faceworkers by Jacobsen et al., only 7% of those starting with 1971 ILO U/C x-ray classification of 0/- or 0/0 (i.e., no pneumoconiosis) had any radiographic progression over a 10-year period (74). Another large study by Reisner from West Germany gave similar results (148); both found the risk of pneumoconiosis to be highly correlated with the amount of dust exposure. Importantly, Jacobsen's study found that CWP in miners with early dust retention (Category 0/1 or 1/0) was more likely to progress than the CWP classified as Category 0/0. In other words, in miners who develop the disease sooner, it progresses more rapidly. Thus the chance of radiologic progression over 10 years at a mean dust concentration of 2 mg/M^3 is essentially zero for a miner with x-ray Category 0/0, but is 20% for one with Category 1/0. Similar differences were found at all levels of dust exposure during the 10-year study; this suggests either a harmful effect from early pneumoconiosis predisposing to more rapid dust accumulation or variability in individual susceptibility to CWP.

The attack rate of progressive massive fibrosis (PMF) has been well-studied in over 100,000 British coal miners by McLintock et al. (106). Calculations from their data show a PMF attack rate of approximately 0%, 1%, 11% and 21% over an eight-year period for simple pneumoconiosis Category 0, 1, 2, and 3, respectively. Rephrased, this means that 1.5% of coal miners with 1971 ILO U/C Category 2 or more simple pneumoconiosis would develop progressive massive fibrosis per year. Independent of this increased attack rate of PMF with increasing simple CWP category, the authors also found an increased attack rate with more rapid progression of simple CWP. This again raises the question of increased individual susceptibility to the effects of coal dust. It is not known whether the given risks of contracting PMF continue indefinitely. Particularly in applying these data to the U.S. mining experience, one should note that substantial regional differences were noted in the above studies which could not be fully explained by the various factors mentioned. In part these differences may be explained by the type of coal mined, dust concentration, and free silica level. A recent report suggests a significant role of free silica content on the attack rate of PMF (72).

While radiologic regression of both simple and complicated CWP can be seen, this apparent reversibility is uncommon and may well represent observer variability in reading the radiographs. There is no doubt that the lesions of complicated pneumoconiosis may contract, but this clearly should not be viewed as clinical improvement. Little evidence exists to suggest any significant resolution of pulmonary impairment caused by CWP. In contrast, it is generally acknowledged that PMF may develop several years after employment ceases, and that once initiated, the process may progress whether exposure continues or not (116). In fact, Cochrane et al. could find no effect of continued dust exposure on the pro-

gression of PMF (25). He analyzed the radiographs of miners and ex-miners with PMF over an eight-year period and found that comparing the measured area of involvement on serial PA chest x-rays gave a more sensitive indicator of change than routine clinical readings. Using the former method, 13% of 579 individuals with all stages of PMF showed either no change or improvement in their x-rays over the eight years, and 87% progressed. Looking at the 341 cases starting with less than 20 sq cm of involved area (i.e., Category A), 45% showed progression over the eight year study period. Importantly, Cochrane also showed that the progression was greater in the younger population. Thus, of the group starting with Category A, 69% of those less than age 45 showed progression while only 32% of the older cases progressed.

Alternatively, with regard to the progression of simple CWP, the work of Jacobsen et al. (74), Reisner (148), and the close correlation between the radiographic category and coal content of the lungs (17) all indicate that stopping coal dust exposure should prevent further progression. Hence, the rationale for transferring miners with simple CWP to low dust areas.

Finally, it is important to note that a miner's x-ray category reflects his dust exposure over his entire working life and that with the new and lower dust standards, the natural history of CWP is changing. The data of Jacobsen et al. indicate that the probability of developing category 2/1 or higher simple CWP after 35 years exposure to a mean dust concentration of 2 mg/ M^3 is near zero (74). Thus, in the present generation of beginning U.S. miners, advanced simple CWP and more importantly, potentially disabling PMF, should become a rare occurrence.

Laboratory Investigations

Pulmonary Function Tests

The comments made regarding the nonspecificity of the respiratory signs and symptoms in coal miners apply equally well to pulmonary function abnormalities. Pulmonary function tests cannot diagnose CWP, but can detect physiologic impairment from whatever cause. Therefore, the physiologic evaluation of a miner with symptoms referable to the chest would be the same as for a nonminer.

Simple CWP

While conflicting series exist, several U.S. studies show that miners have lower ventilatory capacity compared to controls or predicted normal (60)(87)(139). This impairment, although significant, has not been large. Thus, in a large nationwide study of working U.S. bituminous miners, the mean FEV_1, FVC, and FEV_1/FVC ratio were normal or more than 90% of predicted in all categories of simple CWP (121). British and other studies also show conflicting results, most showing no effect or a small decline of function with simple pneumoconiosis (5)(62).

It is also important to note that while an occasional study describes decreasing values in Category 3 (65)(151), the majority find no correlation between the radiographic profusion of simple CWP and ventilatory capacity (23)(121)(126)(127)(139)(146). This lack of correlation is consistent with the involvement of small airways as was shown in the "Pathology" section. These peripheral airways normally contribute only 10-15% to the total airway resistance and thus represent a "quiet" zone of the lungs (107). That is, they must sustain substantial damage before changes will be detected by the relatively insensitive standard spirometric tests such as FEV_1 and FVC. In the case of CWP, this concept of small airways involvement is supported by the analysis of Bates (9) and several studies. Morgan, for example, found small increases in residual volume even in miners with a normal FEV_1 and suggested this represented either focal emphysema or, more likely, increased resistance to flow in the small airway (120). The frequency dependence of dynamic compliance found by Seaton in miners with simple CWP, but without significant large airways obstruction, also suggest small airways disease (156).

While it seems that simple CWP *per se* causes mild small airways disease, coal mining exposure may also cause chronic industrial bronchitis as discussed elsewhere. In a large-scale study of working U.S. miners, Hankinson et al. found chronic bronchitis to be associated with small decreases in flow at high lung volumes during a forced expiratory maneuver (52)(53). This, together with a normal total lung capacity, was interpreted as indicating large airways obstruction due to industrial bronchitis without emphysema. It is apparent, therefore, that the reduction in ventilatory capacity sometimes seen in miners with simple CWP can in part be explained by this concept of industrial bronchitis.

Most U.S. and foreign studies of the dif-

fusing capacity in simple CWP have shown normal or slightly decreased values (30)(31)(82)(157)(162). The last three references also noted that the diffusing capacity and, in Cote's studies, several other tests of lung function were lower in those with the "p" type of opacity compared to those with "q" opacity. The explanation for these interesting differences remains unclear.

Studies of gas exchange in U.S. miners report varying results. Lapp and Seaton studied 51 symptomatic miners who had FEV_1/FVC ratio greater than 70% and found an increased physiological dead space to tidal volume ratio (VD/VT) as had been shown in previous studies (89). The VD/VT returned to normal or near normal with exercise in all simple CWP categories except in the combined Category 3 plus Category A of PMF. The alveolar-arterial gradient for oxygen ($(A\text{-}a)O_2$) was similarly slightly abnormal or in the high normal range. Arterial oxygen saturations were in the low normal range. Rasmussen et al. found similar values for VD/VT, ($(A\text{-}a)O_2$) and arterial oxygen saturation in their series of 192 symptomatic miners, including 158 with simple CWP (146). They found marked hyperventilation and impairment of oxygen transfer with exercise, however, even in those miners with normal spirometry. These results, together with their other findings, including frequent pulmonary hypertension, were interpreted as indicating significant pulmonary vascular involvement and ventilation/perfusion abnormalities, even in the absence of chronic airways obstruction. Rasmussen, in a larger study, reached similar conclusions (145) although both his methods and interpretation have been challenged (43). One can conclude that while gas exchange abnormalities are not uncommon in symptomatic miners with simple CWP, it is not clear that they are of great enough magnitude to explain dyspnea.

The lung mechanics in working U.S. coal miners have been studied in a series of articles by Lapp and Seaton (88), Seaton et al. (156), and Morgan et al. (125). These authors found normal or slightly reduced values for static lung compliance and pulmonary recoil pressure in simple CWP. Frequency dependence of dynamic compliance, often considered a sensitive indicator of small airways disease, was found in 17 of 25 cases of Category 2 and 3 simple CWP, while Category 1 showed minimal decrements and Category 0 showed no significant change in dynamic compliance with increasing respiratory frequency.

Rasmussen's group studied systolic pulmonary artery pressures in 26 symptomatic miners who had no or mild obstruction and found elevated pressures in 7 at rest, and in 18 during exercise (146). In contrast, studies using the more reliable mean pulmonary artery pressure have found substantial pulmonary hypertension to be quite unusual in CWP without airways obstruction (86)(90)(133)(162). Interestingly, Lapp's group found the "p" type of opacity to be more associated with high pulmonary artery pressures. This group also performed lung scans in miners and found perfusion defects in 9 of 21 patients with simple CWP (155). However, in only two cases, both with Category 3 profusion and one probably with silicosis, were the scan defects thought to be due to pneumoconiosis. Abnormalities such as old tuberculosis were found to explain the scan defects in the remaining seven patients.

In discussing simple CWP, it should be noted that anthracite coal seems to cause greater impairment for a given x-ray category than does bituminous (121)(127), although the reason for this is not completely known.

Complicated CWP (PMF)

In contrast to simple CWP, PMF is often associated with abnormalities in most pulmonary function tests and these are generally correlated with the extent of lung involvement. However, this correlation is found primarily in the higher categories of PMF; indeed, several studies have shown pulmonary function to be near normal in Category A (23)(126). With Categories B and C of PMF, one frequently finds a marked reduction in ventilatory function, low diffusing capacity, and gas exchange abnormalities (89). Pulmonary hypertension and cor pulmonale may be present even without severe obstruction. Respiratory failure is not uncommon in severe cases. Depending on the relative proportions of emphysema and fibrosis present, pulmonary compliance may be either increased or decreased. There are few large studies in this area; the available data is reviewed by Marek (101).

Radiological Studies

The chest radiograph is the only way of confirming the presence of CWP in life other than lung biopsy, which is rarely, if ever, indicated.

In general, if the history of exposure and the chest radiograph are consistent with the diagnosis of CWP and any signs and symptoms present are compatible with this condition, no other diagnostic procedures are necessary.

The typical radiographic opacities seen in simple CWP are rounded opacities of the "q" size and shape, although "p" and less commonly "r" opacities are seen (Figures II-32-II-36). A mixed pattern of rounded and irregular opacities are sometimes found. The lesions tend to predominate in the upper zones of the lungs in the earlier stages (2)(137). Amandus et al., found that approximately 6% of working U.S. coal miners showed small irregular opacities either alone or with rounded opacities on their radiographs (1). The irregular lesions were correlated with cigarette smoking, as well as bronchitis, age, and years worked underground. Lyons et al. have shown that these lesions (unlike rounded opacities) also correlate with the extent of emphysema and the impairment of FEV_1 (99).

The lesions of PMF may vary greatly in shape as well as size, and may be single, multiple, unilateral, or bilateral (Figure II-37). They usually predominate in the upper lung zones but can occur anywhere; they may cavitate and (rarely) calcify. Typically, they are multiple irregular masses that tend to migrate towards the hila by contraction of fibrotic tissue. PMF usually develops on the background of Category 2 or 3 simple CWP, although traction by the conglomerate masses may overdistend the remaining tissue, rendering the simple CWP less evident on x-ray. Thus previous radiographs are often important in supporting the diagnosis of PMF.

It is important to note that none of the described radiographic features of CWP is pathognomonic. In addition, certain findings should cause one to question this diagnosis. These would include noncalcified hilar or mediastinal adenopathy and pleural effusion. Caplan's syndrome—the occurrence of multiple pulmonary nodules in a miner who usually has rheumatoid arthritis with subcutaneous rheumatoid nodules —is mentioned as the one variant of CWP which progresses rapidly. These lesions resemble necrobiotic rheumatoid nodules pathologically and may appear over a period of weeks as opposed to years for PMF.

Lung Biopsy

While this procedure can usually confirm a diagnosis of CWP (see Pathology section, page 353), it is rarely medically indicated. The main clinical setting in which a biopsy may be necessary is when a solitary mass lesion is seen on the chest radiograph. Here one may not be able to distinguish carcinoma (or other mass lesion) from PMF, and biopsy may therefore be indicated for proper patient management.

Other Tests

While much interesting research has been done in the area of other laboratory (particularly immunological) tests, at present no laboratory test is diagnostic for CWP, and none can accurately predict which miner will develop the disease.

Treatment

No effective treatment of CWP is known. While previous animal studies have suggested polyvinylpyridine-N-oxide (PVNO) is effective, although less so than in silicosis, a recent controlled long-term trial by Weller in monkeys with simulated CWP showed no beneficial effect of the chemical (175). At present the clinician's role in respiratory treatment is limited to managing the complications of CWP or the incidental cardiopulmonary diseases which afflict the miner, to providing guidance as to further occupational explosure, and to strongly advise against smoking.

CWP and Tuberculosis

Tuberculosis plays a doubtful role in producing PMF, as discussed in "Pathology." While there is little evidence indicating any increased risk of contracting tuberculosis in CWP, some articles suggest that CWP affects the pathogenicity of the infection. These studies show antituberculosis chemotherapy to be less effective in the presence of CWP (11)(44)(143). While silicosis clearly predisposes to tuberculous disease which may then be poorly responsive to chemotherapy, it is unclear if the small amount of silica in coal mine dust causes a similar problem. Although standard antituberculosis chemotherapy should be adequate when *Mycobacterium tuberculosis* is identified in the sputum, some cases will have to be individualized (116). Thus, the clinical, bacteriologic and radiologic

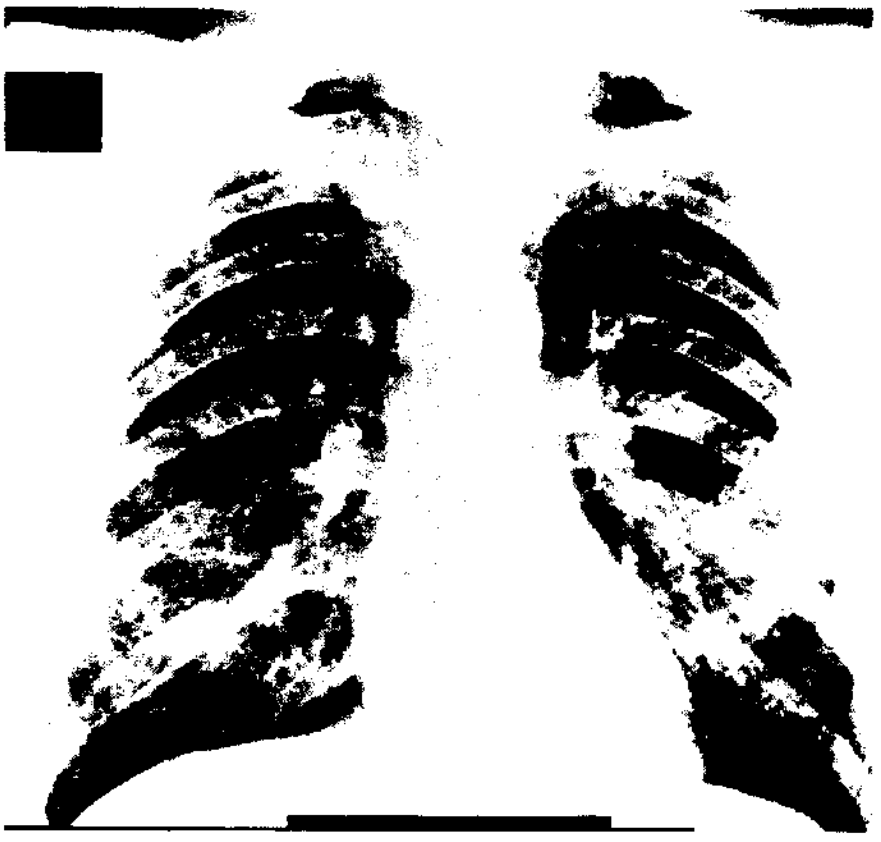


Figure II-32. Normal chest radiograph. Profusion category 0/0.

response of miners with historical (roofbolters or drillers) or radiologic (eggshell calcifications) evidence of high silica exposure should be carefully followed. In this regard, Dubois et al. have found rafampin-combined chemotherapy to be effective in new and retreatment cases of TB in coal miners (37).

DIAGNOSTIC CRITERIA

The usual clinical criteria for diagnosing coal workers' pneumoconiosis are a documented history of substantial (usually at least ten years) exposure to coal dust and a chest radiograph consistent with the diagnosis (ILO 1980 Classification Profusion Category 1 or greater). The combination is not pathognomonic, and consideration must be given to other occupational as well as nonoccupational chest diseases. Usually with a history, physical exam, and old chest x-rays, diseases which can present with a pattern mimicking simple CWP, such as miliary tuberculosis, histoplasmosis, or sarcoidosis are easily differentiated. Some other pneumoconioses, particularly silicosis, can present with an identical radiographic pattern. In such cases a lung biopsy is the only way to obtain a definitive diagnosis, but is not recommended as a substitute for a good occupational history.

Differentiating other diseases from complicated CWP is more difficult. The diagnostic possibilities may include tumor, tuberculosis, fungal diseases, and some vasculitic conditions. As in the case of simple CWP, a lung biopsy should yield a definitive diagnosis in clinically confusing cases; however, in most cases this procedure is not required for proper patient management.

A more complete list of the differential

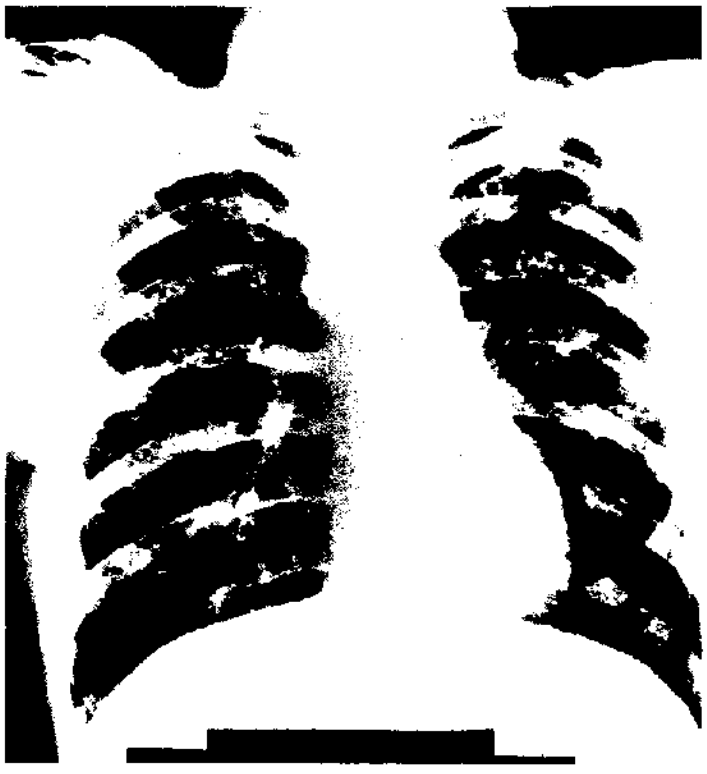


Figure II-33. Simple coal workers' pneumoconiosis. Profusion category 1/1. Size and shape r/r.

Source: (69)

diagnositic possibilities in CWP is presented in standard textbooks and in articles by Van Ordstrand (170) and Pendergrass et al. (140). Since the occupational history and chest radiograph are not pathognomonic, there is always room for some debate regarding the diagnosis in coal workers' pneumoconiosis. The Department of Labor has recently established standards for the assessment of lung impairment and disability under the Black Lung Benefits Reform Act of 1977 (167). These guidelines, and the arguments for and against them, are contained in the preamble preceding these standards. It should be emphasized that these criteria were formulated to facilitate the processing of compensation cases and are not meant to substitute for a good clinical evaluation of any medical problem in a miner.

PREVENTION

As noted previously, the basic methods to prevent coal workers pneumoconiosis and associated airways disease were defined over a century ago when the importance both of adequate mine ventilation and removal of affected miners was appreciated. With definition of the dose-response relationship between respirable coal mine dust and pneumoconiosis, new dust standards were quickly adopted in Great Britain and the United States. The U.S. dust standard was initially set at 3 mg/m^3 to be reduced to 2 mg/m^3 by 1973. As shown in Table II-23, the U.S. coal mining industry has made excellent progress in meeting the dust standard with over 90% of U.S. mining sections now in compliance. Smaller mines and long-wall operations, which are increasing in number, tend to have greater difficulty meeting the standard. Dust control has been achieved by attention to mine ventilation and assisted by the use of water spraying on the continuous miner (a mining machine).

Use of diesel powered mining equipment may offer safety advantages, but introduces possibly hazardous exposures (oxides of nitrogen and other irritating gases as well as carcinogens and mutagens) into the mining environment. The

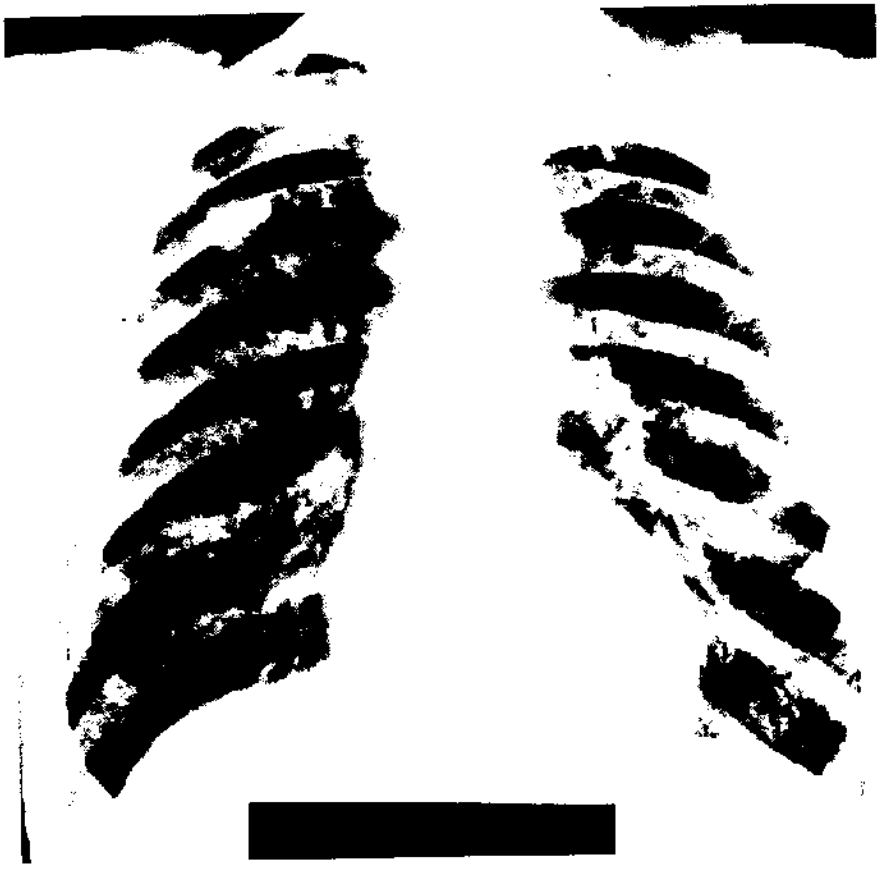


Figure II-34. Coal workers' pneumoconiosis. Profusion category 2/2. Size and shape p/p.

extent of these exposures and their possible effects are not yet adequately defined. Although significant free silica concentrations in U.S. mines are usually relatively low, those who drill through siliceous overburdens may get high exposures to free silica and develop acute silicosis. Therefore, sampling for free silica in addition to respirable dust is always necessary.

Medical surveillance is the second important means to prevent disabling pneumoconiosis. The Federal Coal Mine Health and Safety Act of 1969 mandated pre-employment and periodic medical examinations be offered to underground coal miners through a program to be administered by NIOSH. The Act also provided that these examinations be paid for by the mine operator and that miners with evidence of coal workers' pneumoconiosis be given the opportunity to transfer to a low dust area (1 mg/M^3 or lower) without loss in pay (transfer rights and rate retention). The National Coal Workers' Health Surveillance Program was established in 1970 by NIOSH at its Appalachian Laboratory for Occupational Safety and Health (ALOSH). Under regulations adopted by NIOSH, medical examinations (occupational questionnaire and PA chest radiograph) are conducted by facilities (hospitals and clinics) certified by NIOSH and located throughout the coal fields (168). Qualified physicians ("A" Readers) located at the facilities interpret the radiograph for clinical pathology and for pneumoconiosis according to the 1980 ILO Classification scheme. Radiographs are then sent to ALOSH where they are coded and batched for a second reader ("B" Readers—those who have passed a NIOSH proficiency examination on interpretation of the pneumoconiosis). If "A" and "B" readers do not agree within one subcategory of the 1980 ILO Classification, further "B" readings are obtained until agreement is achieved. Miners with Category 1 profusion are judged to have evidence of pneu-

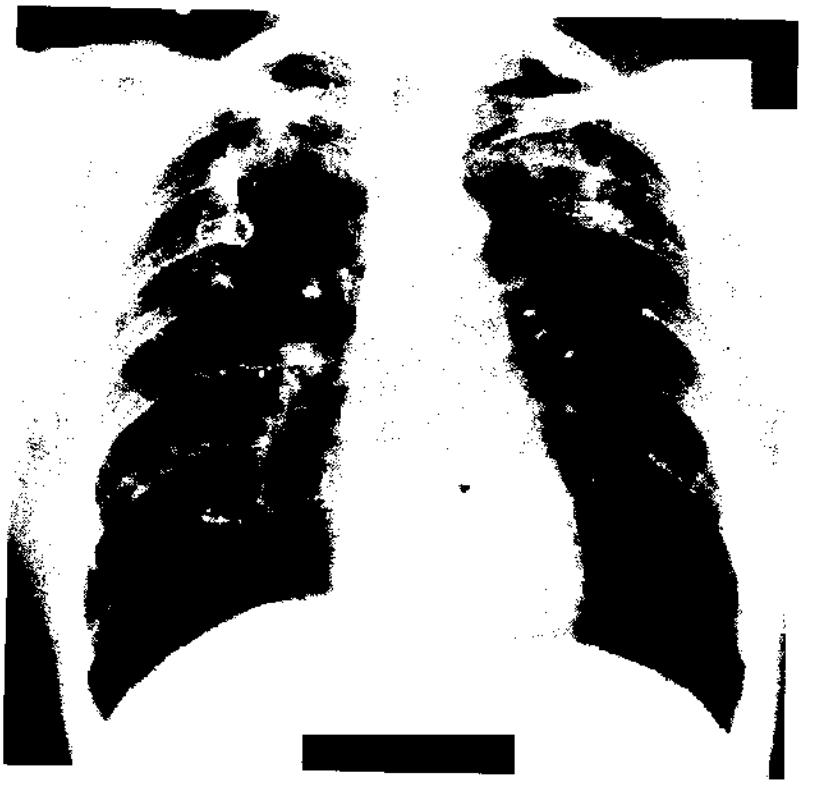


Figure II-35. Simple coal workers' pneumoconiosis. Profusion category 2/2. Size and shape q/q.

Source: (69)

moconiosis and are extended an option to transfer to a low dust area through a letter from the Administrator of MSHA. MSHA continues to follow miners who have exercised their option to ensure exposure to low dust levels.

Results of the third round of examinations of the National Coal Workers' Health Surveillance Programs are shown in Table II-24. The prevalence of CWP continues to slowly decline with most advanced simple CWP and PMF occurring among miners with more than twenty years underground. This, therefore, largely reflects previous higher dust exposures. Based on the British dose-response experience and current trends in dust control and medical surveillance findings, it appears that *advanced CWP per se* should become a relatively uncommon condition among U.S. coal miners.

Airways obstruction is now a much more important problem among coal miners than is pneumoconiosis. Because of the dose-response relationship between coal mine dust exposure and decline in FEV_1, NIOSH has proposed that lung function testing be incorporated into the surveillance program. This has not yet been adopted and as a result, reasonably good prevalence and incidence estimates will continue to be available for CWP but not for lung impairment. However, based on available epidemiological information and current respirable coal mine dust levels, the contribution of dust exposure to decline in lung function should be reduced. Without a similar decrease in the consumption of cigarettes, cigarette smoking will assume an even larger role in causing airways obstruction among miners. Unfortunately, the dispute over the role of coal dust and smoking has polarized the miners and operators and, to an extent, the public health community, making it difficult to convince miners of the importance of cigarette smoking in the causality of their lung disease. This remains the area of greatest con-

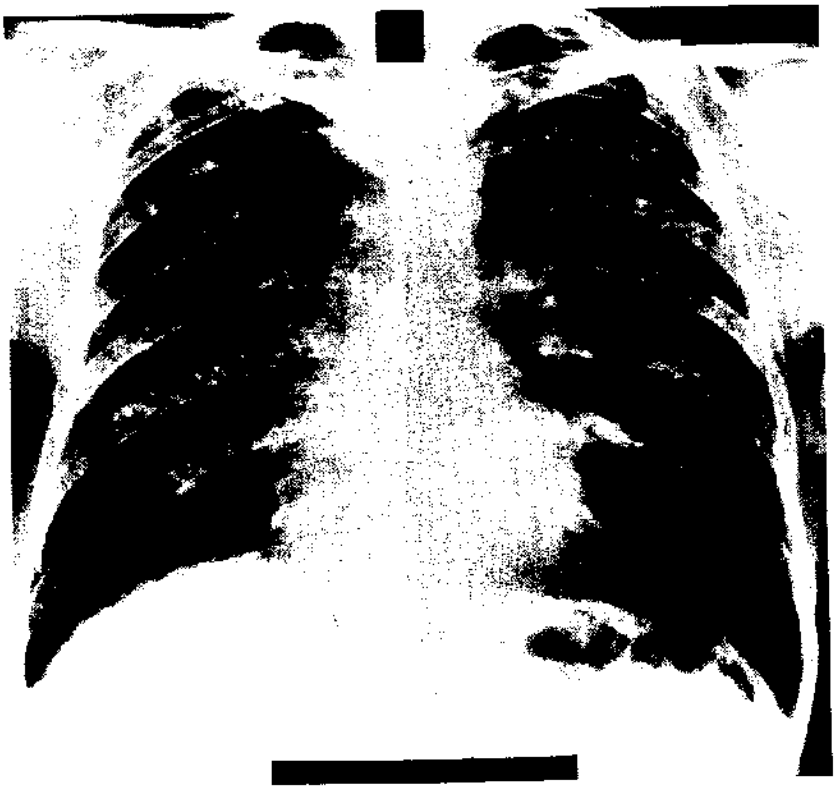


Figure II-36. Simple coal workers' pneumoconiosis. Profusion category 3/3. Size and shape r/r.

cern in prevention of respiratory disease of coal miners.

RESEARCH NEEDS

Despite the extensive research reviewed in the previous sections, a number of important questions remain partially or fully unresolved:

1. Pathological/epidemiological investigations on inflated lungs together with other clinical data and good occupational and smoking histories are needed to document the nature and extent of chronic bronchitis and the emphysemas among coal miners with and without simple CWP and PMF.
2. Prospective epidemiological studies of coal miners should continue with an emphasis on further defining dose-response relationships and risk factors relating to airways obstruction at low levels of coal mine dust exposure (under 2 mg/M^3).
3. Development of more sensitive and specific methods to detect dust deposition in the lung should continue to be a research priority.
4. Further refinement of epidemiological methods to reduce variability in testing miners should continue to be a research priority.
5. Cohort, case-control, and laboratory investigation is needed to resolve etiologic questions regarding the role of coal mine dust exposure and other potential risk factors in stomach cancer incidence.

Figure 11-37. Coal workers' pneumoconiosis—progressive massive fibrosis category C.

Source: (69)

6. Epidemiological and experimental assessment should be continued to clearly define whether diesel emissions pose a hazard to miners, and if so, the nature and extent of the health effects and measures which might be taken to mitigate or prevent such possible health effects.

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