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Respiratory Disease in Coal Miners

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Coal miners develop a variety of lung diseases as a result of their workplace exposures. Of these, coal workers' pneumoconiosis (CWP) has received the most attention because of its clear occupational association. Bronchitis and emphysema resulting from coal mine dust exposure, clinically indistinguishable from their nonoccupational analogues, are also prevalent and are associated with significant morbidity among coal miners. The group of lung diseases for which miners are at increased risk have been called "black lung" in coal mining communities and in U.S. federal compensation legislation. To date, most scientific investigations and preventive efforts have been directed toward the control of CWP.

Although a disease attributed to coal dust inhalation was reported following the autopsy of a Scottish miner in 1831 (1), the nature of coal miners' lung diseases was debated for the next 150 years. CWP was not recognized as an entity distinct from silicosis in Great Britain until approximately 1940 (2,3). Coal mining has been an important industry in the United States since the early 19th century, but official recognition that coal mine dust causes chronic lung disease, premature disability, and death did not occur until the final third of the 20th century. The disastrous Farmington, West Virginia, mine explosion and fire, in which 78 miners died, combined with findings of U.S. Public Health Service studies and a significant level of political activism among coal miners (4) led to the passage of the Federal Coal Mine Health and Safety Act of 1969. This Act (5), amended in 1977 (6), directs the Secretary of Labor to set standards for exposure to toxic materials so that "no miner will suffer material impairment of health or functional capacity even if such miner has regular exposure to the hazards dealt with by such standard for the period of his working life." Specifically, health standards for exposure to coal mine dust were established with the intent "to permit each miner the opportunity to work underground during the period of his entire adult working life without incurring any disability from pneumoconiosis or any other occupation-related disease." A respirable coal mine dust standard (3 mg per m^3 air, later reduced to 2 mg per m^3) was established. The Act also provided for rigorous inspection procedures, medical examinations for working miners and autopsies for deceased miners, a federally administered compensation program for miners with disabling lung diseases, and federal agency right of entry for research to advance understanding of the health effects of mining.

The United States has extensive coal deposits (Fig. 22.1). Owing to the increasing scarcity and cost of petroleum as a fuel, coal will continue to be an essential energy source. It is impossible to extract coal without some dust exposure, so it is critical to understand the relationships between coal mine dust exposure and the development of respiratory diseases in order to diagnose, treat, and prevent them.

EPIDEMIOLOGY

CWP is a well-defined medical entity resulting from the deposition of coal mine dust in the lung and the reaction to the deposited dust resulting in coal macules, coal nodules, and progressive massive fibrosis (PMF). Because of the nonspecific nature of chronic obstructive pulmonary disease (COPD) and the frequent concurrent presence of multiple risk factors, such as dust exposure and cigarette smoking, the diagnosis of lung diseases related to coal mine dust has led to disagreement and

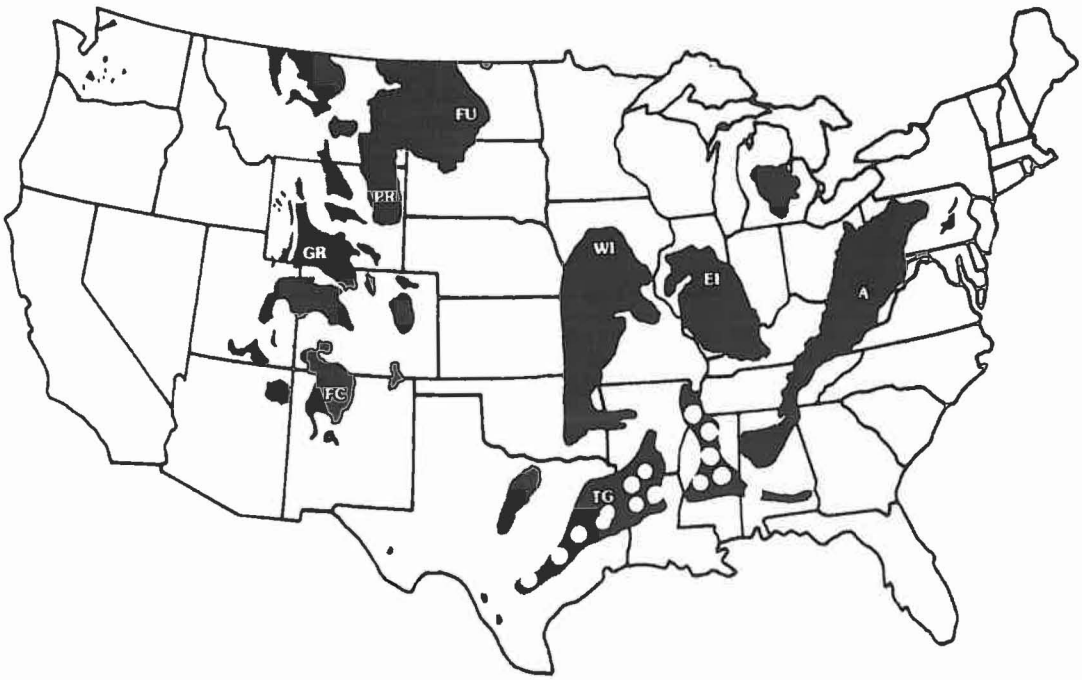

Figure 22.1 Coal deposits in the U.S. mainland. *Completely filled areas* have coal deposits; *partially filled areas* have scattered coal deposits. A, Appalachia; EI, eastern interior; WI, western interior; TG, Texas Gulf; PR, Powder River; FU, Fort Union; GR, Green River; FC, Four Corners.

controversy over the definition and diagnosis of black lung (7). Ongoing epidemiologic, pathologic, and clinical studies have provided important information, helping to resolve some of these questions.

Perhaps more investigation has been directed toward lung diseases of coal miners than toward any other occupational disease. Meiklejohn, for example, cited more than 100 reports and articles published before 1950 on the health of coal miners (8–10). These early works helped to reveal the extent and nature of respiratory disease in miners but were unable to quantify the effects of coal dust because they lacked reliable exposure estimates. Analyses of large epidemiologic studies with comprehensive exposure measurement components, such as the British Pneumoconiosis Field Research (11), have permitted the establishment of exposure-response relationships for a number of medical conditions. The results of these have been applied to the setting of dust control standards throughout the world. In addition, these intensive multifaceted studies have clarified many aspects of the causation and significance of lung disease in coal miners.

Morbidity, Coal Mine Dust Exposure, and Other Risk Factors

Coal Workers' Pneumoconiosis

Studies undertaken between 1960 and 1970 in various regions of the United States (12–18) revealed varying degrees of risk of CWP with years worked underground. These findings contributed to a movement toward the imposition of restrictions on respirable dust levels in U.S. coal mines, culminating in the 1969 Federal Coal Mine Health and Safety Act (5). The resulting reductions in dust exposure have led to a substantial drop in CWP prevalence rates (Fig. 22.2), although evidence of continuing overexposure continues to be reported (19).

British epidemiologic studies of the relationship between prevalence and incidence of CWP and environmental measurements have consistently revealed that the predominant adverse exposure factor is respirable mixed coal mine dust (20–24). Coal rank (age and hardness of the coal) has also been found to play a role, in that risk increases with the carbon content of the coal. Quartz (silica), on the other hand, was found to be a minor contributor to CWP development in general, although the environmental levels were low on average (20,21). However, quartz was implicated in a study of a group of cases of unusually rapid progression of simple pneumoconiosis (25). In addition, miners with a particular form of PMF that appeared to consist of conglomerations of the larger nodules of simple CWP (radiographic type *r* opacities) had received higher exposures to quartz than had their controls (26). The risks to coal miners of even brief overexposures to silica dust have been re-emphasized (27). Exposures to rock dust containing silica were implicated in the development of pneumoconiosis in a study of Spanish coal miners (28).

Among nonoccupational factors, smoking was not found to affect simple CWP development (29) nor did

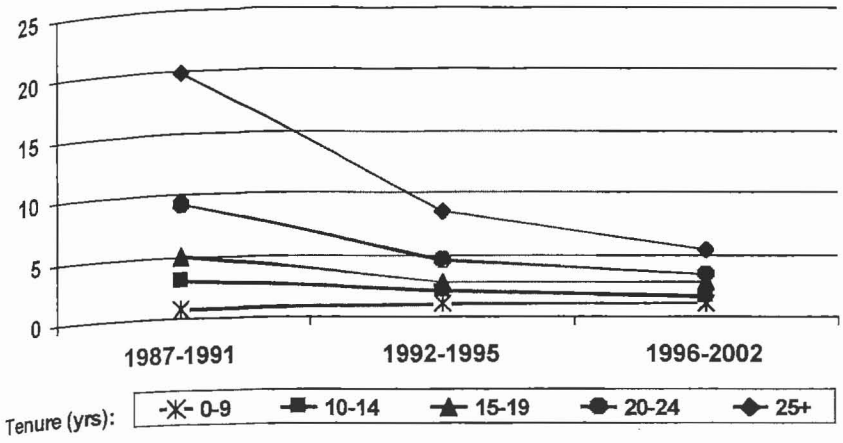

Figure 22.2 Trends in CWP prevalence by tenure among examinees employed at underground coal mines, U.S. National Coal Workers' X-Ray Surveillance Program, 1987 to 2002.

bronchitis appear to play a role (30). However, an important risk factor for the development of PMF is the presence of simple CWP (23,31,32), and risk increases with increasing category of small opacity profusion on the radiograph. There are also indications that body mass and breathlessness may be positively related to future development of PMF (33). [The latter result is in agreement with pathologic findings that show widespread emphysema in miners with PMF (34).] No other important factors have emerged, and considerable unexplained variation remains in the data. A detailed study of eight "anomalous" mines (i.e., ones with much higher or much lower rates of simple CWP than expected for measured dust levels) was able to account for only part of the variation in five and for none in the remaining three (35).

The current coal mine dust exposure limit for underground coal mines in the United States relies substantially on estimates of exposure-response relationships for CWP obtained from study of British miners (20). Figure 22.3 shows the relevant curve, which relates the estimated 35-year risk of category 2 or higher small, rounded opacities to the concentration of respirable mixed coal mine dust. Category 2 was chosen as the response following the advice of Cochrane, who, noting that the incidence rate of PMF increased markedly among miners with simple CWP of category 2 or higher, argued that the logical way to control the appearance of PMF is to concentrate on preventing miners from reaching category 2 of simple pneumoconiosis. Since the curve predicts zero incidence of category 2 or greater at 2 mg per m^3, that dust concentration was adopted as the federal standard.

More recent British analyses of exposure and response have concentrated on PMF as the response variable (24) (Fig. 22.4). These findings were derived from a study of 52,264 5-year intervals of risk for more than 30,000 British miners. Clear exposure-response relationships were found for dust and coal rank, the predictions of risk being somewhat greater than those from previous analyses. Although these investigations confirmed that the risk of contracting PMF increases with category of simple CWP, they also showed that PMF could develop over 5 years in miners whose initial chest radiograph was normal. This risk, which appeared to be dependent on the degree of previous dust exposure, implies that PMF could not be eliminated exclusively through control of simple CWP.

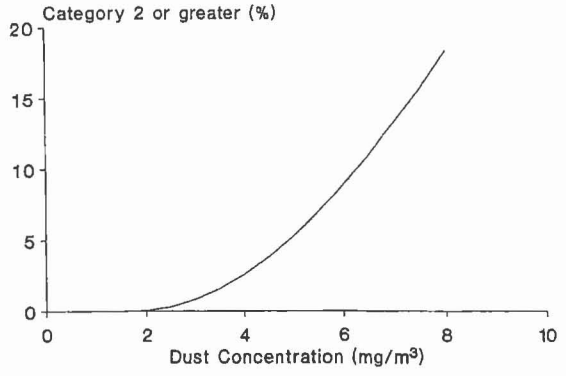

Figure 22.3 British miners' predicted risk of contracting category 2 or greater small, rounded opacities over 35 years plotted against dust concentration.

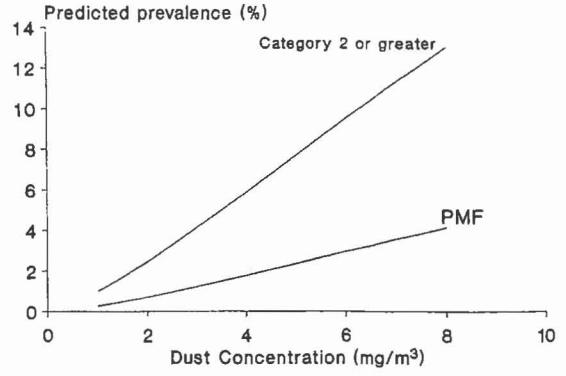

Figure 22.4 British miners' predicted risks of contracting category 2 or greater small, rounded opacities and PMF after 40 years of work plotted against dust concentration.

Exposure-response relationships have been reported among miners from countries other than Britain. Reisner (36,37) has demonstrated clear exposure-related effects of dust and coal rank in German miners. German studies of exposure-response indicate a risk between 1% and 14% for category 2 or greater over 35 years at 4 mg per m^3 and <4% for 2 mg per m^3 (38).

Results from South African coal miners showed a prevalence of 4.2% category 1 or greater (maximum reader) for an average dust exposure of 56.8 mg-years per m^3 (equivalent to an average concentration of 1.62 mg per m^3 over 35 years) (39). In the United States, dust concentration data collected in the past few years before the reductions due to federal regulation were used to compute retrospective dust exposures for miners working at Round 1 of the National Study of Coal Workers' Pneumoconiosis (40) and then applied to estimation of exposure-response for CWP (41). Clear effects of dust exposure, coal rank, and age on prevalence of CWP (small, rounded opacities and PMF) were seen. The results are shown in Figure 22.5, which provides predicted prevalences of category 1 or greater, category 2 or greater, and PMF for a range of dust exposures. These findings were confirmed in another study involving U.S. underground coal miners and ex-miners medically examined between 1985 and 1988 (42). They tend to be somewhat higher than those derived from studies of British, German, and South African coal miners (23,38,39).

Most of the studies noted previously were undertaken on working miners and were therefore vulnerable to bias induced by ignoring miners in poor health who left mining, such as that identified in older, higher dust-exposed miners (43). Attfield and Seixas (42) showed that miners who reported leaving work because of their health had higher levels of abnormality than their colleagues who remained at work. However, a study of exposure-response in groups of ex-miners and current miners led the authors to conclude that "estimates of risk of simple pneumoconiosis in relation to exposure to mixed respirable dust in working miners adequately describe the relation found in men who have been miners but have left the industry" (44). A similar observation was made regarding the development of PMF in current and ex-miners (45). (These observations should be contrasted with those for ventilatory function discussed later, for which evidence indicates more extreme effects of dust on those who leave coal mining.)

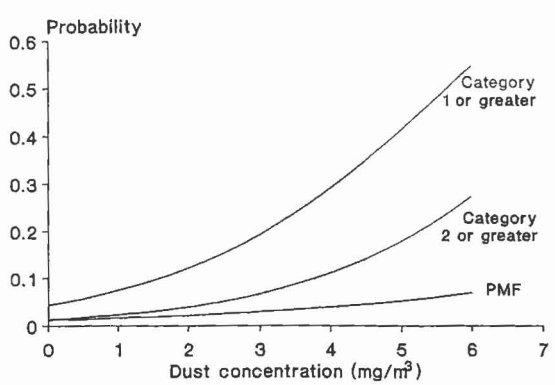

Figure 22.5 U.S. miners' predicted risk of contracting category 1 or greater small, rounded opacities; category 2 or greater small, rounded opacities; or PMF after 40 years of work in coal mining.

Although most exposure-response assessments for CWP have employed a logistic type of model using cumulative exposure as a predictor, some information exists on alternative approaches. The findings of Reisner (36) support the concept of a "residence time" effect, an observation confirmed to some extent by Hurley et al. (22). In an exploration of modeling approaches, Attfield et al. (43) found little evidence for a threshold in exposure-response for CWP. Rather, a nonzero prevalence at zero exposure was indicated, consistent with a background level of detected abnormality probably due to diseases other than CWP, as well as artifactual causes of chest radiographic abnormalities.

In summary, the main environmental factors involved in the development of simple CWP are coal mine (mixed) dust exposure and coal rank. Age, quartz exposure, and dust residence time probably also play a role, although these effects appear to have secondary importance unless silica levels are high. Category of simple CWP remains a strong predictor of PMF development, but the occurrence of PMF has been found to be related to dust in the absence of simple CWP. The recently developed exposure-response relationships indicate that CWP incidence for dust levels of 2 mg per m^3 air or less may be greater than was predicted in the past.

Other Lung Diseases

Correlation of measured dust exposures with indicators of lung disease other than pneumoconiosis has consistently revealed clear relationships with both respiratory symptoms and lung function. In the first of such studies, associations were found between the prevalence and incidence of respiratory symptoms and dust levels for workers who had never smoked and for those who currently were smokers, but this was apparent only for the younger miners (46). Findings for U.S., Australian, German, and Sardinian coal miners support these observations (47–50).

Cross-sectional studies of forced expiratory volume in 1 second (FEV_1) undertaken in a number of countries have consistently shown it to be inversely related to cumulative dust exposure (or work in coal mining) after allowance is made for age, height, and smoking history (44,47,51–57). In general, results from these studies have revealed dust exposure effects comparable to those due to smoking. Dust exposure effects have been seen among current, former, and nonsmokers, but smoking

was not found to potentiate the effect of dust. After adjustment for dust exposure, no additional effect of the presence of CWP on FEV_1 was noted. Some suggestion of a greater dust exposure effect among ex-miners has been reported (44). Ex-miners in this study did have lower overall ventilatory function than current miners.

Several studies have shown that forced vital capacity (FVC), like FEV_1, is inversely related to dust exposure and that both lung function variables tended to decline somewhat in parallel. Some have suggested that this implies that dust-induced lung damage has a different physiologic basis than that due to smoking. Despite the tendency to parallelism, an inverse relationship between the FEV_1:FVC ratio and dust exposure has also been reported (52), although the association was weaker than those for FEV_1 and FVC.

Findings from a joint analysis of ventilatory function and respiratory symptoms among British miners led the authors to conclude that both smoking and dust exposure can lead to clinically important respiratory dysfunction (58). Logistic models fitted to responses based on reports of persistent cough and phlegm, reduced FEV_1 and cough and phlegm, FEV_1 less than 80% of predicted value, and FEV_1 less than 65% of predicted value all showed significant relationships to dust exposure. The prevalences of the four responses at high dust exposures were found to be similar to that among smokers who had hypothetically zero dust exposure (Fig. 22.6).

Longitudinal changes in ventilatory function in coal miners have also been linked with dust exposure or coal mining work (28,50,59–64). However, the relationship is complex and varies depending on the age or, more likely, prior mining tenure of the miners (64). New miners appear to suffer a fairly severe initial decline after beginning work in mining (64–66). This loss then ameliorates, but it is still detectable in experienced miners (60,61). In a study of young miners by Carta et al. (50), annual decline in FEV_1 was significantly related to concurrent dust exposure but was inversely associated with prior dust exposure. This effect might be attributed to worker self-selection, whereby those able to withstand the higher dust exposures remain in dusty jobs. Support for this is found in the study by Petsonk et al. (67), which showed that miners with greater airway responsiveness were less likely to work in dusty jobs.

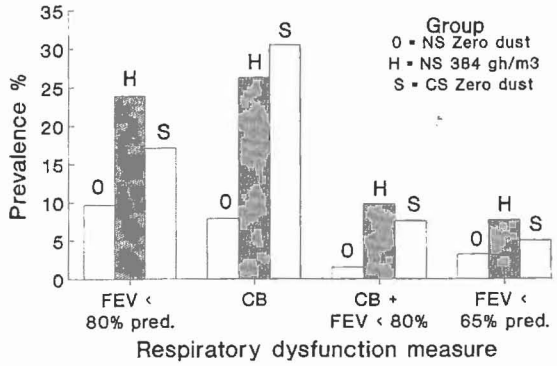

Figure 22.6 Predicted prevalence of four indices of respiratory dysfunction for never smokers (NS) at 0 and 384 gh/m^3 air and current smokers (CS) at 0 gh/m^3.

The general effect of dust exposure on FEV_1 seems to occur at a loss of approximately 0.7 ml of FEV_1 per gram-homs per cubic meter (gh/m^3) (approximately 5 ml per year for a dust exposure of 4 mg per m^3—twice the current U.S. compliance level). Although it is tempting to dismiss this apparently small effect of dust exposure, it must be remembered that it is an average effect being observed in a relatively healthy population fit enough to work in an arduous job. If one closely examines the average effect of smoking reported in the various studies on coal miners, it will be seen that the coefficients are also small (e.g., approximately 5-mL loss per year per pack smoked) (68). Although this decrement is also apparently of little clinical importance, it is known that smoking is a major cause of lung disease. Hence, rejection of an effect just because its average magnitude is not clinically significant can be misleading. In a study of the distributional pattern of ventilatory function related to dust exposure and smoking, the effects appeared to be similar (42). Furthermore, a study of British miners identified a group of ex-miners who apparently had suffered a severe response to their dust exposure (44,69). The authors concluded that this indicates that dust exposure can give rise to severe respiratory impairment in the absence of PMF. Accelerated declines in ventilatory function were associated with increased mortality after adjustment for smoking (70). Lastly, in an analysis of autopsy material on coal miners, Leigh et al. (71) found a clear inverse relationship between the extent of emphysema and FEV_1 in both smokers and nonsmokers, whereas emphysema and lung dust content were positively related. In total, these results suggest that dust-related loss in FEV_1 is not of trivial significance.

In contrast to the study of exposure-response for CWP, in which extensive exploration of various dust composition parameters (e.g., coal rank and silica content) has taken place, investigation of exposure-response for other lung diseases has concentrated almost exclusively on mixed mine dust exposure. This has resulted from lack of any convincing evidence concerning coal type or dust composition, confirmed by Coggon et al. (72), who showed wide variations in the proportionate mortality ratio (PMR) for CWP across Britain (mainly trending with coal rank) but a relatively uniform excess PMR for chronic bronchitis and emphysema over the different coalfields. However, there may be risk factors associated with coal mining but not with coal dust exposure, including exposure to contaminated dust-suppressant water, to gases from explosives, and work involving roof bolting (73).

In contrast to the wealth of information available on CWP, bronchitis, obstructive lung disease, and emphysema in coal miners, little attention has been paid to other lung diseases. In this respect, there is one case report concerning a miner reported to have occupational asthma due to *Rhizopus nigricans* (74). Mold colonies, including *R. nigricans* and other genera, were found extensively in the mine in which the miner worked.

In summary, clear relationships between various measures of ventilatory function and dust exposure have been found in various unrelated groups of underground coal miners. For miners working where dust levels are not well-controlled, there is evidence that some may experience nontrivial affects to their health. Fortunately, steps taken to reduce dust exposures for prevention of CWP are expected to have similar efficacy in preventing nonpneumoconiotic diseases (75).

Mortality, Coal Mine Dust Exposure, and Other Risk Factors

Mortality patterns among coal miners have been studied systematically for approximately 100 years, beginning with British occupational mortality statistics for 1890 to 1892. These studies have generally shown increased standardized mortality ratios (SMRs) for accidents, respiratory disease, respiratory tuberculosis, and stomach cancer. Overall (all-cause) SMRs have varied somewhat, from a high of 195 in an early study (76) to levels slightly above 100 in a number of more recent large analyses (77–80). The later studies reveal evidence of healthy worker selection effects, with some reports showing statistically significant elevations in SMRs for ex-miners and current miners who likely changed to surface jobs because of ill health.

Standardized rates for death from pneumoconiosis have consistently shown increased risks associated with PMF (79–86). Most of the studies, however, failed to detect any elevation in mortality risk associated with the presence of simple CWP. Miller and Jacobsen (80) found that simple CWP was associated with a 2% or 3% reduction in 22-year survival rates, but there was no apparent association with the category of simple CWP. They also reported that the relative risk of death over a 22-year period for miners who developed PMF while young (ages 25 to 34 years) was 3.5 compared to those without CWP.

Elevated death rates due to lung disease other than CWP have also been reported. Rockette (77) found increased rates of emphysema, influenza, asthma, and tuberculosis, whereas Miller and Jacobsen (80) concluded that miners exposed to excessive amounts of respirable dust are at elevated risk of death from chronic bronchitis or emphysema. An SMR of 426 has been reported for nonmalignant respiratory disease among Dutch coal miners (87). In two studies examining mortality in association with quantitative measures of cumulative dust exposure (80,88), mortality from pneumoconiosis increased with extent of dust exposure.

Lung cancer rates typically have been low (89), and no obvious relationship of lung cancer mortality with dust exposure has been detected (90). An excess of deaths due to stomach cancer has been seen among miners in Britain (78,91), the United States (77,92), Holland (87,93), and Japan (94). There is a suggestion that stomach cancer risk is related to exposure to coal mine dust (80). Ames (95) describes a number of hypotheses that could explain the higher gastric cancer mortality among coal miners. Among these is Meyer's hypothesis, which postulates that stomach cancer incidence would be greater in miners with good lung clearance since the dust deposited in the lungs would be transported more effectively from the lungs and then swallowed (96). Some findings of Dutch coal miners support this view (93), although evidence for this hypothesis was not found for U.S. coal miners (90).

Surface Coal Miners

Compared to the sizable number of studies and reports on underground coal miners, surface miners have been relatively neglected. This may be due to the generally lower dust exposure they experience and, until recently, their fewer numbers. Despite their lower level of occupational dust exposure in general, severe disease can and has occurred in surface miners. Also, many coal miners often spend time working in both surface and underground mines and thus, experience dust exposure from both sources.

Studies of U.S. surface coal miners have revealed an overall level of respiratory disease that is considerably lower than that seen generally in underground coal miners (97,98). In an initial study (97), 4% of the workers showed signs of CWP, but only seven of 1,438 had radiographic category 2 disease or worse. Obvious signs of excessive lung disease other than CWP were not seen. However, the impression that work in strip mining was relatively benign was upset by a subsequent report of acute silicosis among a surface miner working in rock drilling operations (99). The hazards of drilling were fully revealed in a later study in which almost half of the cases occurred in the minority of miners who had worked in drilling (98). In addition, the drillers' disease was more severe and included one case of PMF. These findings confirmed that overexposure to quartz remains a hazard for surface coal miners.

RADIOGRAPHY

A diagnosis of CWP is generally based on chest x-ray findings combined with an occupational history of significant coal mine dust exposure. The chest radiograph,

however, appears to be an insensitive tool for detecting CWP compared to tissue pathology (25% sensitivity when macules only are recorded; 40% for macules or mild degrees of micronodules), although its specificity appears to be good (90% specificity for 10 films with no observed pathologic abnormalities related to CWP) (100,101). Other dust diseases of miners are usually undetectable by plain chest radiography (102). Therefore, this discussion of radiography applies only to the use of plain films in the recognition of CWP. Other diagnostic methods must be employed to identify the other diseases related to coal mine dust exposure.

Coal mine dust can produce a pattern of chronic interstitial fibrosis, most often nodular but frequently mixed nodular and irregular, and occasionally exclusively irregular. However, a radiograph with predominantly irregular opacities in a coal miner raises the possibility of previous exposure to occupational hazards such as asbestos or of a nonoccupational interstitial lung disease. In simple CWP, radiographic small opacities vary in size from 1 mm to 1 cm. Fibrosis is usually noted first in the upper or middle lung zones and predominates there, but potentially it is visible anywhere. As disease progresses, opacities may be found throughout the lung fields. Smoking and aging are thought to influence opacity profusion, particularly irregular opacities (103), although interstitial opacities are rarely found in healthy workers in the absence of dust exposure (104,105).

The differential diagnosis of the small opacities typical of simple CWP includes (a) diseases that produce acute nodular lesions, such as miliary tuberculosis and viral pneumonia; and (b) diseases that produce chronic nodular patterns such as other pneumoconioses, metastatic disease, tuberculosis, and other granulomas. Silicosis typically presents with the same radiographic pattern as CWP and so can be differentiated only by occupational history or pathologic examination.

By radiographic convention, nodular opacities 1 cm or larger are defined as PMF. The large opacities of PMF almost invariably occur on a background of simple CWP. They are usually rounded, may be multiple, and are most often found posteriorly in the upper lung zones. PMF may affect an entire lobe (Fig. 22.7). The large opacities of PMF must be differentiated from malignancies, granulomatous diseases, and other less common causes.

In order to improve recognition and reporting of CWP and other pneumoconioses, international standards have been adopted (106). These classification methods have contributed materially to epidemiologic studies of coal miners, medical surveillance programs, and clinical assessment of CWP.

The need for consistency and accuracy in the interpretation of chest radiographs for surveillance purposes led to the development of a program of training and certification of readers by the National Institute for Occupational Safety and Health (NIOSH). Trained readers who pass a competency examination are designated B readers. Their classifications are generally more consistent with one another than those of non-B readers, and they are given more weight in both epidemiologic investigations and legal proceedings. Nevertheless, there remains significant inter- and intrareader variability, particularly with mild disease (107–109).

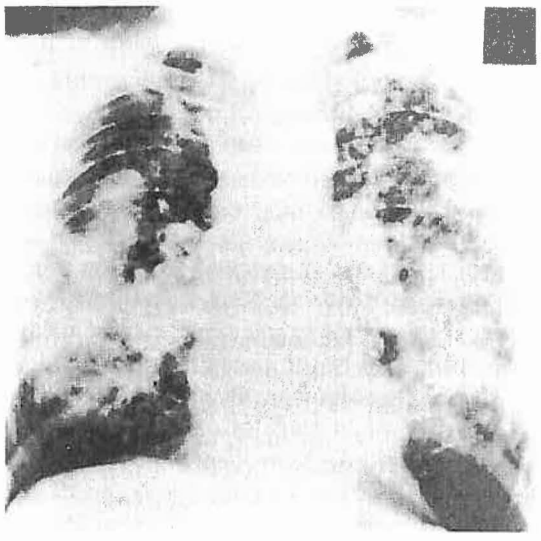

Figure 22.7 PMF, category C, of coal worker's pneumoconiosis.

More technologically advanced imaging methods such as high-resolution computed tomography (HRCT) have been promoted by some for the diagnosis of coal workers' lung diseases (110–112). To date, there is no standardization of interpretation of the changes identified on standard CT or HRCT. Classification systems have been proposed but not yet validated (113,114), and epidemiologic investigations have not been sufficient to establish relationships between levels of exposure to coal mine dust and CT abnormalities (115,116). There is substantial discordance between findings on HRCT and the chest radiograph, with the HRCT more sensitive to low levels of abnormality (117). CT and HRCT should be utilized when there are specific clinical indications or when they are part of a research protocol; routine use for surveillance of miners is not indicated.

PATHOLOGIC FEATURES

The descriptive work of Heppleston (118) and Gough et al. (119) helped define the characteristic pathologic features of CWP as an entity distinct from silicosis. With the widespread practice of fixing lungs in inflation for investigation has come better definition of the pathologic changes in CWP. It is now generally recognized that the primary lesion of CWP, the coal macule, occurs specifically among workers exposed to coal mine dust,

regardless of geographic location, rank of coal, or type of dust. This characteristic lesion of CWP has been defined by the Pneumoconiosis Committee of the College of American Pathologists as follows: "A focal collection of coal dust-laden macrophages at the division of the respiratory bronchioles that may exist within alveoli and extend into the peribronchiolar interstitium with associated reticulin deposits and focal emphysema" (120). Other lesions specific to coal mine dust exposure are the well-recognized nodular lesions of simple CWP, PMF, and Caplan's lesions. Emphysema and bronchitis resulting from coal mine dust exposure do not have pathognomonic lesions that identify the occupational cause.

Macrophages laden with coal mine dust are found free in the alveoli of anyone who inhales coal mine dust (Fig. 22.8). The coal macule is similar in appearance to dust macules found in urban dwellers and smokers, but coal macules are more profuse. Macules range in size from 1 to 5 mm and may be rounded, irregular, or stellate. The coal macule is typically found together with a 1- or 2-mm zone of enlarged air space referred to as focal emphysema (Fig. 22.9). Histologically, the coal macule consists of dust-laden macrophages that surround the first-, second-, and third-order respiratory bronchioles, extending into alveoli and interspersed with fine reticulin and a variable amount of collagen. Since the lesion may occur with other occupational and environmental exposures (e.g., graphite and carbon black), it is important to identify the nature of the dust particles (121–123). Bituminous and anthracite coal can usually be distinguished by light microscopy (124).

The nodular lesions of CWP have been classified as micronodules (up to 7 mm diameter) and macronodules (7 to 20 mm diameter) (120). These lesions are almost invariably seen on a background of coal macules, are usually rounded black lesions, and may be surrounded by enlarged air spaces. Nodules, unlike macules, are firm to palpation. They are usually found in the region of the respiratory bronchiole and may coalesce to form PMF. Histologically, nodules consist of dust-laden macrophages in a stroma consisting of collagen and reticulin (Fig. 22.10). Degenerative changes, including calcification, cholesterol crystallization, blood vessel obliteration, and infarction, are commonly observed.

The pathologic definition of PMF is arbitrarily based on the diameter of lesions. The Pneumoconiosis Committee of the College of American Pathologists recommended a minimum diameter of 2 cm for morphologic investigations (120) despite the radiographic definition of PMF as nodules 1 cm or greater in diameter (125).

PMF lesions may be unilateral or bilateral, often in the upper and posterior regions of the lung. They may be rounded or irregular and frequently cross lobar fissures. The lesions tend to retract toward the hilum, and in so doing, they destroy blood vessels and airways and greatly distort lung architecture (Fig. 22.11). The larger PMF lesions tend to cavitate, sometimes discharging a black liquid into a communicating airway and resulting in symptomatic melanoptysis. Histologically, the tissues are composed of bundles of irregularly arranged collagen or reticulin, coal dust, and coal dust-engorged macrophages. Less collagen is found toward the center

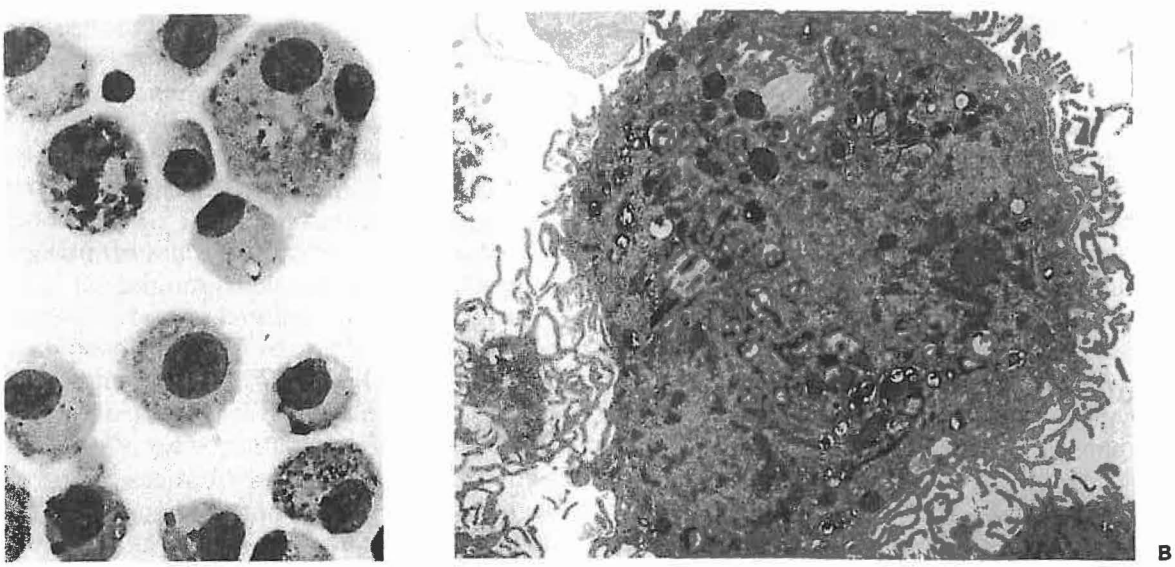

Figure 22.8 Coal-laden macrophages recovered by bronchoalveolar lavage (BAL) from an active coal miner. **A**: Variably shaped dark particles in light microscopic study of alveolar macrophages (400×). **B**: Transmission electron microscopy study of alveolar macrophage from a coal miner shows particles in phagolysosomes (4,200×). (Courtesy of T. Takemura, MD, and V. Ferrans, MD)

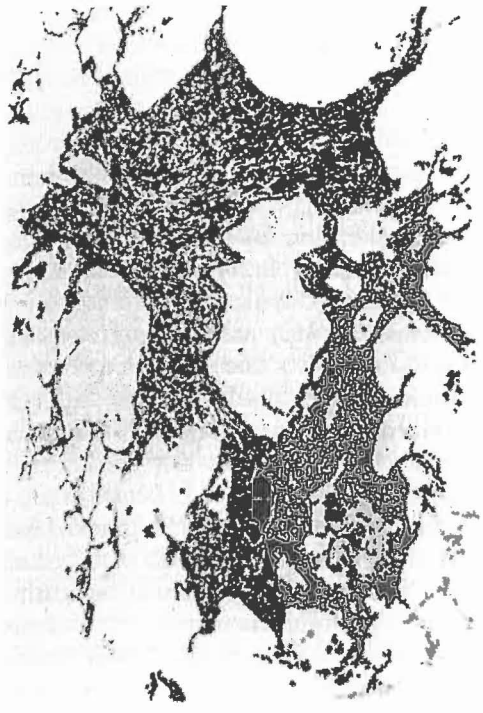

Figure 22.9 The coal macule surrounding a dilated respiratory bronchiole.

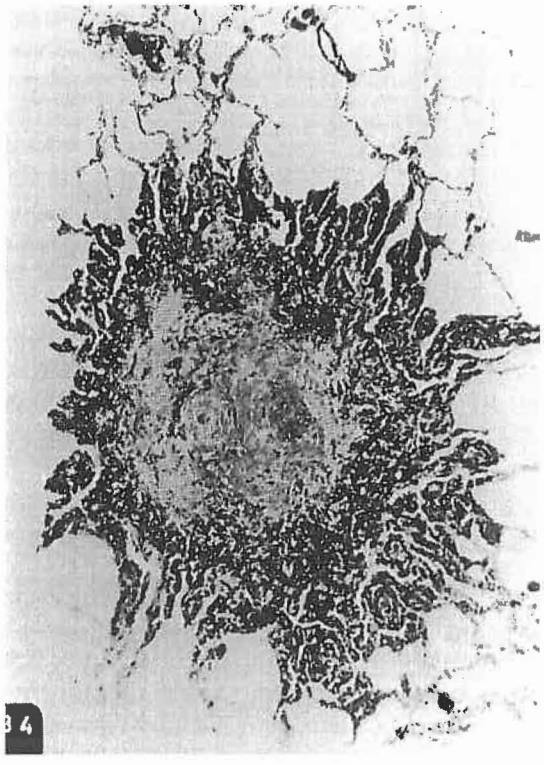

Figure 22.10 A macronodular lesion of a coal worker's pneumoconiosis has a center that is becoming hyalinized.

of the lesions, and an obliterative vasculitis is observed in peripheral areas (126).

Rheumatoid pneumoconiosis (Caplan's syndrome) is characterized by large (1 to 5 cm diameter) nodules that typically have smooth rounded borders, concentric internal lamination, and (relative to PMF lesions) little coal dust. The characteristic histiocytic palisading and necrobiosis found in most rheumatoid nodules is usually peripheral and focal.

Silicotic nodules are frequently found in coal miners' lungs and arise from respirable free silica exposure, usually a reflection of the silicaceous rock surrounding the coal seams. These nodules are usually found incidentally in conjunction with coal macules and nodules. They are typically rounded and firm, and they have smooth borders and pale centers that are relatively free of coal dust. They also tend to coalesce, forming PMF or conglomerate silicosis. Histologically, silicosis nodules have characteristically concentric lamination of collagen fibers about a hyalinized center (Fig. 22.12). A study of 3,365 autopsies of U.S. underground coal miners revealed that approximately 12% had classic silicosis nodules. A relationship was seen between tenure in mining and prevalence and severity of silicosis. In addition, job category and geographic locality were important determinants of silicosis prevalence (127).

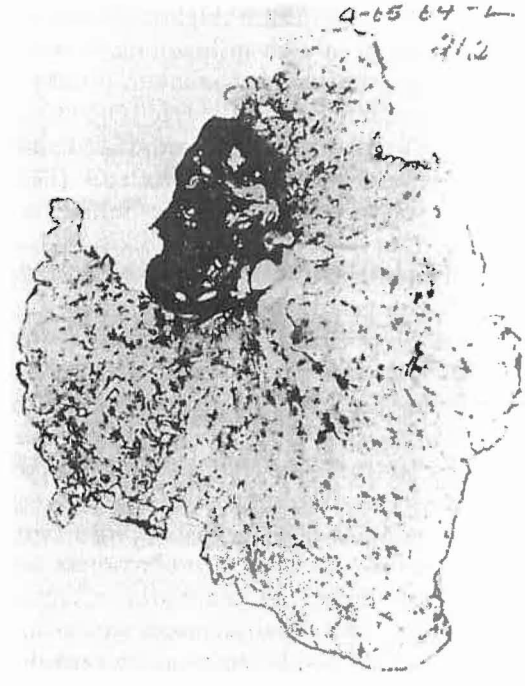

Figure 22.11 This whole lung section shows PMF of a coal worker's pneumoconiosis against a background of nodules, macules, and focal emphysema.

The relationship between pathologic findings, the weight and composition of dust retained in the lungs, and radiographic information has long interested researchers. Early studies showed that there was a clear and direct link between the weight of dust in the lung and the radiographic category of pneumoconiosis (128–130). Weight for weight, the mineral (noncoal) portion of the dust appeared to be more responsible for

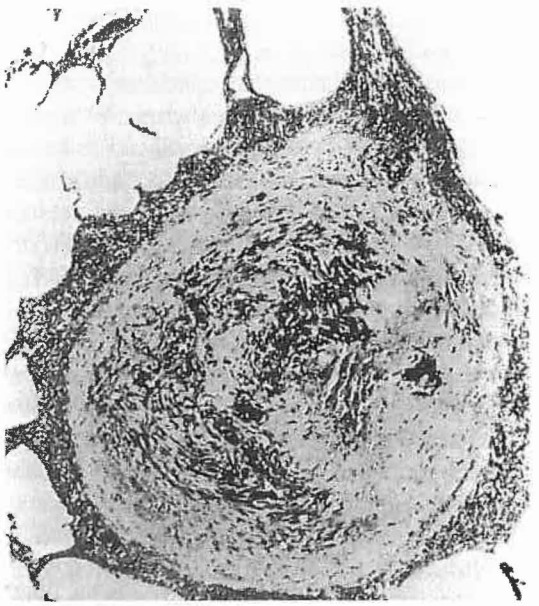

Figure 22.12 Silicosis nodule with paracicatricial emphysema in a coal worker's lung.

the radiographic opacities than the coal fraction. This implies that miners in high-rank coal mines would have to retain appreciably more dust in their lungs to attain the same radiographic classification (the ratio of coal to ash in the mixed mine dust in high-rank mines is usually much greater than that in low-rank mines). Since lung collagen content did not relate to opacity profusion after adjustment for lung dust (129), it was suggested that radiographic changes are related simply to dust accumulation (130).

Additional work has supported the link between the amount and composition of retained dust and opacity profusion on radiographs (131). It also revealed that the relationships between radiographic data, lung dust content, and lung disease are more complex than was originally supposed. In this respect, it is clear that the size of the opacity must be taken into consideration. In both high and low coal rank areas, miners with the smallest rounded opacities (*p*) had greater lung dust weights than miners with the largest rounded opacities (*r*), after the data were controlled for degree of profusion (131). An explanation for this may be that type *p* opacities may be the manifestation of the summation effect of very many small dust deposits, whereas larger opacities are often individually radio-opaque. The relationship between lung dust weight and profusion of opacities was much more evident among those miners with predominantly type *p* opacities than for type *q* (medium-sized small, rounded opacities) or *r* (132). For the type *r* opacities and a high proportion of ash in the retained dust, no association between profusion and dust weight was found (132), although the number of cases was small.

A detailed study of the pathologic lesions of pneumoconiosis and opacity type showed that lungs of miners with predominantly type *p* opacities contained mostly dust macules and pinhead nodules (smaller than 1 mm diameter but offering resistance to a needle) (132). For these miners, the only pathologic lesion that correlated with radiologic profusion was the number of pinhead nodules; in fact, total lung dust correlated even better. Opacity profusion in miners with predominantly type *q* opacities related best to numbers of small nodules (1 to 3 mm diameter), although nodules 3 to 9 mm in diameter also played a role. In these cases, lung dust weight correlated weakly with profusion. In the few type *r* cases, association between profusion and the largest size nodules was poor, as was that with dust content.

These findings support the view that profusion of type *p* opacities is a reflection of numbers of macules and very small nodules, and that it relates mostly to coal dust deposition per se, particularly for high-rank coals. Type *q* and *r* opacities are seen in miners with the larger lung nodules. The presence of these types of small opacities seems linked more to the ash content of the dust than to the coal fraction; the correlation between radiographic profusion and weight of dust is less clear. The possibility that the dust associated with coals of different rank gives rise to different disease processes is suggested by radiographic findings (26) and pathologic data (133). A study undertaken to compare radiographic and pathologic appearances of PMF provided some support for this hypothesis (134).

There is a clear correlation between dust exposures and retained dust in the lung (135,136), between retained dust and the presence and severity of pathologic lesion (135,136), and between dust exposure and the presence and severity of pathologic lesion (136). Pathologic abnormality was better predicted by retained dust than by dust exposure (136). Miners with PMF appeared to have retained more dust per unit of exposure than those without PMF (135). With regard to dust composition, coal miners from high-rank mines had been exposed to low levels of ash and had low levels of ash in their retained dust. In contrast, miners of low-rank coal had higher levels of ash in the retained dust, with the ash content being greater proportionately than it was in the mines (135). It is not clear how this excess occurred, although differential deposition or retention is an obvious hypothesis.

Pathologic studies of emphysema in different groups of coal workers have repeatedly shown an excess over levels in controls (137–139). Much discussion has centered on the implications of this finding. Questions center around the nature, cause, and significance of the emphysema and on the potential for bias in the selection of coal miner cases in some of the studies. Recent work has clarified some of the outstanding issues (34,140,141).

Not only was the excess of emphysema among coal workers confirmed in a study that controlled well for bias (34) but also emphysema was found to be related to both dust retained in the lungs (34,131) and pathologic measurements of pneumoconiosis (131). The finding that miners with PMF also have elevated amounts of emphysema unrelated to the PMF lesions (34) is consistent with the epidemiologic finding that dyspneic miners are at greater risk of PMF development (33). Importantly, the presence of emphysema was related to dust exposure during life (131), particularly the coal rather than the silica component (141), and to FEV_1 percent predicted (71,141). These findings, therefore, indicate a causal relationship between dust exposure and emphysema and the potential for ensuing disability (142). There is some indication that irregular opacities on chest radiographs are associated with pathologic signs of emphysema and interstitial fibrosis (34).

Despite the clear association between occupational exposure and chest symptoms reported by Rae et al. (46), results from the few pathologic studies of bronchitis have been mixed. Leigh et al. (141) found no evidence of an association between bronchial mucous gland:wall ratio and years worked underground, even though other studies of Australian miners had shown correlations between gland:wall ratio and various symptoms and signs of bronchitis (143,144). However, a study using measured dust exposures rather than the surrogate years underground (145) found that maximum mucous gland:wall ratio did correlate with lifetime occupational exposure. Douglas et al. (145) said of their results, "[They] lend support to the view that irritants encountered in an occupational environment may play an important part in the development of hypersecretion of mucus."

DISEASE MECHANISMS

The development and progression of pulmonary disease induced by coal mine dust inhalation has been linked to the following mechanisms of damage, inflammation, and lung scarring: (a) direct cytotoxicity, (b) activation of oxidant production by alveolar macrophages, (c) stimulation of inflammatory factors, and (d) stimulation of fibrogenic factors (146–148).

Coal mine dust has been reported to cause cell membrane damage directly, as measured by hemolysis of red blood cells, lactate dehydrogenase release from alveolar macrophages, and lipid peroxidation (149–151). It has been proposed that such cytotoxicity is related to trace metal contamination of the coal dust. Christian and Nelson (152) reported that water leachates of Pennsylvania coal are more potent in inhibiting mammalian cell growth in culture than those from Utah coal. This potency difference is directly related to the nickel content of these coal samples and to the prevalence of CWP in miners from these two states. Alternatively, the cytotoxicity of coal dust may be related to its ability to generate radicals. Lebeder et al. (153) reported that crushing coal results in the generation of surface free radicals. Pennsylvania coal has been shown to be more potent in generating radicals than Utah coal (154). In addition, anthracite coal generates more radicals upon grinding than bituminous coal, which relates to the greater hemolytic activity of anthracite coal samples (155). Although direct cytotoxicity from coal dust has been reported in vitro, it is significantly less than that observed with quartz. Indeed, the in vitro toxicity of coal dust is significantly enhanced by the addition of 10% quartz (150). In contrast to in vitro evidence of the cytotoxicity of coal dust, inhalation of coal mine dust (2 mg per m^3, 7 hours per day, 5 days per week for 2 years) failed to increase markers of lung damage (bronchoalveolar levels of protein or lactate dehydrogenase) in a rat model (156). However, inhalation of coal dust in rats did cause lipoproteinosis, with the magnitude of response being related to coal rank (157).

Coal dust has been shown to increase the production of reactive oxygen and nitrogen species by alveolar macrophages in both a rat model and coal miners. Inhalation or intratracheal instillation of coal dust in rats resulted in increased ruffling and spreading of alveolar macrophages harvested by bronchoalveolar lavage (BAL) (156). This was associated with an increase in reactive oxygen and nitric oxide-dependent particle-induced chemiluminescence from alveolar macrophages (156,158). Coal dust exposure also induced mRNA for inducible nitric oxide synthase in these BAL cells. In general, this oxidant production in the rat model was less in response to coal dust than to silica exposure (158). Similar results have been reported for alveolar macrophages harvested from coal miners. Increased surface ruffling has been associated with coal mining (159,160). Although enhanced production of reactive oxygen species was not reported in asymptomatic coal miners (160), enhanced superoxide and hydrogen peroxide production by alveolar macrophages was reported in miners with CWP (161,162). Increased nitric oxide-dependent chemiluminescence has also been reported in alveolar macrophages harvested from coal miners (163). The magnitude of reactive species production by human alveolar macrophages was directly related to the severity of CWP (160–162).

Exposure of rats to coal dust has been reported to increase the number of alveolar macrophages and neutrophils harvested by BAL (164) as well as the number of blood and interstitial monocytes (165). A similar increase in the number of lavaged alveolar macrophages was reported in miners with CWP but not asymptomatic coal miners (159–162). This recruitment of

inflammatory cells is associated with the stimulation of the production of chemotactic and inflammatory mediators in response to coal dust exposure. In vitro exposure of alveolar macrophages to coal dust resulted in enhanced release of platelet-activating factor (166), prostaglandin E_2 and thromboxane A_2 (167), tumor necrosis factor-α (TNF-α), interleukin-6 (IL-6) (168), and IL-1 (169). In vivo exposure of rats to coal dust has been linked with increased production of leukotriene B_4 and thromboxane A_2 by alveolar macrophages (170). TNF-α in acellular BAL fluid was found to be decreased in asymptomatic miners compared to control volunteers (171). However, elevated BAL fluid levels of TNF-α, IL-1, and IL-6 were noted in miners with category 2/2 CWP (171,172). Increased production of TNF-α was also noted from alveolar macrophages and from blood monocytes harvested from miners with CWP (173,174), Likewise, increased production of IL-1, IL-6, and monocyte chemoattractant peptide-1 (MCP-1) from alveolar macrophages has been reported in miners with CWP (173,175,176).

Coal dust exposure has also been associated with elevated production of fibrogenic factors. As noted previously, TNF-α production is elevated in macrophages from coal miners with severe CWP and PMF (171–174). TNF-α is an initiating cytokine that has been correlated with the release of chemoattractants and fibroblast growth factors (177,178). Indeed, treatment of mice with antibodies to TNF-α has been shown to effectively suppress pulmonary fibrosis (179). IL-1 production is also elevated in coal miners with severe CWP or PMF (171–173). Evidence indicates that IL-1 can directly stimulate collagen synthesis (180). Furthermore, knockout of the IL-1 gene resulted in substantial mitigation of silica-induced pulmonary lesions in a mouse model (181). In addition, the fibrogenic factors fibronectin (161,171), alveolar macrophage-derived growth factor (161), and transforming growth factor-β (161,182) are elevated in BAL samples from coal miners with CWP. Evidence indicates that production of these fibrogenic factors is directly related to disease severity (182,183). Elevated fibrogenic factors associated with CWP include competence factors, such as platelet-derived growth factor, and progression factors, such as insulinlike growth factor (182,183).

In conclusion, evidence supports the following mechanism of initiation and progression of CWP and PMF in coal miners: Coal dust can directly generate reactive species as well as stimulate oxidant production by alveolar macrophages. These oxidants not only cause lung injury but also can activate transcription factors, such as nuclear factor-kappa B and activator protein-1, which induce messenger RNA for the production of a host of chemokines, inflammatory cytokines, and growth factors associated with the formation of lung lesions and fibrosis (184).

PULMONARY PHYSIOLOGY

Cross-sectional and longitudinal epidemiologic investigations, reviewed previously, have provided most of the information available concerning pulmonary changes resulting from exposure to coal mine dust. A limited number of clinical investigations have extended our knowledge in this area. Studies of coal miners have consistently shown relatively high prevalences of dyspnea, chronic bronchitis, and COPD in addition to CWP. Pathology investigations demonstrate elevated levels of emphysema in miners (185). Coal mine dust exposure and cigarette smoking have both been found to be unambiguous risk factors for airways obstruction among coal miners, although they do not appear to be synergistic.

No single pattern of pulmonary response has been identified that uniquely differentiates the physiologic derangement of miners from that of others without coal mine dust exposure. Some confusion has resulted from studies comparing lung function among coal miners with radiographic evidence of simple CWP to that of miners without CWP (52,186). These studies attempt to correlate two different effects of coal mine dust exposure: (a) development of CWP, and (b) changes in pulmonary function. Since both groups studied have had significant coal mine dust exposure, it is not surprising that such studies often show little or no difference in pulmonary function tests (15,187). Nevertheless, a matched sample analysis of a large number of U.S. bituminous coal miners, regardless of category of simple CWP, has shown that both bronchitis and cigarette smoking are significant factors that influence both lung volumes and flow rates (188). FEV_1 and FEV_1:FVC ratio were found to be the measures that best discriminated between smoking and nonsmoking groups and between groups with and without bronchitis. PMF is associated with significant decrease in lung function, particularly in categories B and C of that disorder (189,190). Coal mine dust exposure per se has been associated with accelerated loss of FEV_1 in longitudinal studies and with reduced FEV_1 in both cross-sectional field studies and laboratory investigations.

Studies of lung volume in coal miners have revealed a slight increase in total lung capacity among obstructed and nonobstructed miners (191). Those without airways obstruction have been found to have consistent increases in residual volume, which tended to increase with the radiographic category of CWP. Similarly, studies of dynamic compliance, believed to reflect narrowing or closure of small airways, found that most nonobstructed miners who had category 2 or 3 CWP had significant decreases, as did some category 0 or 1 subjects (186). The clinical significance of these early physiologic changes is not clear.

Diffusing capacity has been found to be reduced among those with predominately type *p* opacities of

simple CWP, among those with category B or C PMF, and among smoking miners. Nonsmoking miners have generally been found to have diffusing capacity measurements within the normal range (192–194).

Among working miners, resting arterial blood gas tension (PaO_2) has generally been found to be within the normal range or minimally reduced. Miners with airways obstruction tend to show lower PaO_2 during exercise than those without obstruction (195). Significant decreases in PaO_2 with exercise have been found among miners with PMF (196). Nonsmoking miners have been found to have a lower PaO_2 and higher alveolar–arterial oxygen difference than nonsmoking nonminers, both at rest and with exercise (57).

CLINICAL EVALUATION AND MANAGEMENT

Evaluative Examinations

Because there is a federal compensation and benefits program directed exclusively toward miners with lung disease, patients may seek care initially in order to determine their eligibility for these benefits. It is, therefore, important that physicians understand not only the disease process and its diagnosis and management but also the provisions of state and federal law that apply in these cases (7). Physicians who provide information to disability or benefits systems should determine the specific type of information required and provide it, if possible, or indicate how the information might be generated. Ultimately, nonphysicians make administrative decisions concerning benefits eligibility utilizing the physician report in addition to other information. It is, therefore, critical that all reports be complete and accurate. A carefully elicited and recorded occupational and medical history can be invaluable in planning care, ascertaining progress, and ensuring fairness and consistency in benefits eligibility determinations. Examiners should inquire not only into current work but also into past jobs and the reasons for job change. People may switch jobs when poor health precludes continuing their normal occupation. This may be a significant fact as a judgment is made during federal black lung benefits determinations as to whether the applicant was capable of performing his or her usual coal mine job.

A history of chest infections or chest trauma may be important. Medical records, especially previous chest radiographs and pulmonary function assessments, are often helpful. The comparative risk from other hazardous respiratory tract exposures (e.g., asbestos and silica) in the home or in prior workplaces should be evaluated. Smoking is a major risk factor that must be fully defined in terms of time of onset, duration, amount, type, and manner of smoking and (if discontinued) time and reason for stopping. A subjective measure of dyspnea, cough, and phlegm is useful. Questioning what kinds of recreational or avocational activities may have been modified as a result of progressive dyspnea, and the timing of these life changes, is often illuminating.

Dyspnea is the symptom that most often correlates with respiratory impairment. It may be graded via standard questions published in the American Thoracic Society (ATS) Epidemiologic Standardization Project Questionnaire (197), which also contains questions aimed at assessing cough, phlegm, and wheezing. (The ATS questionnaire is currently undergoing revision and validation and will eventually be accessible through the ATS Internet site.) The ATS questionnaire is designed to be administered by a trained interviewer, such as an office nurse, and provides a very useful basis for further questioning of the patient. The questionnaire does not provide information on the relationship between symptoms and mining or other exposures, nor does it permit characterization of nonrespiratory symptoms. It affords no insight into the consequences of dyspnea (e.g., the need to abandon activities such as hunting). Also, unless the subject is questioned specifically, frequent consequences of dyspnea, such as sexual dysfunction, will be overlooked. It is important to thoroughly explore cardiovascular symptoms and signs such as chest pain, orthopnea, ankle swelling, rapid weight gain, and nocturnal dyspnea.

The evaluative examination provides an opportunity to broadly examine the health of the miner and plan future interventions. For example, since occupationally induced hearing impairment, musculoskeletal trauma, and dermatitis are common in coal miners, it is reasonable to assess these systems carefully.

The physical examination should be thorough but with a focus on pulmonary, cardiac, and musculoskeletal function. The examiner should seek evidence of coughing and note whether the patient produces phlegm (if so, the nature of the specimen should be noted). The patient's breathing pattern, breath sounds, and respiratory rate should be observed and recorded. The cardiovascular examination should include inspection for neck vein distention and pulsation; palpation for the presence of a right ventricular lift or heave; and auscultation for determination of the pulmonic closure sound, variation of heart sounds with respiration, and gallop rhythm. If present, liver distention or pulsation and pedal edema should be noted.

A clinical assessment of hearing ability is important; audiometric testing is suggested if diminished capacity is suspected. Special attention should be given to assessment of joint and muscle function and to any evidence of trauma.

Laboratory investigations should include, at a minimum, posteroanterior and lateral chest radiographs, spirometry, and a hematocrit determination. An

electrocardiogram may also be useful. The chest radiographs should be interpreted in light of the history and physical examination. If possible, previous radiographs should be obtained and evaluated together with the current radiograph using the current International Labour Office classification to assess pneumoconiosis while paying particular attention to other thoracic abnormalities (106). The electrocardiogram should be evaluated if exercise testing is contemplated, if there is an irregularity of heart rhythm, or if right heart strain is suspected.

Spirometry is the single most important test in evaluating a miner's lung function. Test procedures and published standards are available and have been incorporated into black lung disability determination standards promulgated by the U.S. Department of Labor (198). The FEV_1 is the most useful measurement of the spirogram. It is reasonably reproducible, less effort dependent than the FVC or maximal voluntary ventilation, and has proved to be the test that correlates best with severe impairment and mortality. The FVC and ratio of FEV_1 to FVC are also important, but they are dependent on a full and reproducible FVC. The maximal voluntary ventilation is a difficult test to perform, particularly for patients with significant impairment; however, it remains part of some standard disability determination protocols.

It should be borne in mind that these tests may be influenced by intercurrent infection and the use of bronchodilators. Some miners have a reversible component to their airways disease. In these cases, repeat spirometry following bronchodilation may be of some benefit in planning clinical interventions. An improvement of 10% to 15% supports a trial of bronchodilators; however, many clinicians opt for a "clinical trial" in the absence of such data. Spirometry results should be compared with available population standards, one of which is incorporated into the current federal black lung standards. Since predicted values vary with age, gender, height, and race, these factors should be taken into consideration when interpreting results (195,199,200). Also worth considering is that working people as a rule are healthier than the average person when they begin employment (201). It is, therefore, not unexpected for miners to be cognizant of a loss of exercise capacity, even when their spirometry values do not fall below an arbitrary level of abnormality. Comparison of results of a current spirometry examination with one performed in the past can give some indication of relative loss over time (202). Some analyses have highlighted the fact that impaired workers may have more variable spirometric findings than healthier ones (203). All available measurements for a miner should be assessed, and care must be taken not to ignore information merely because it does not meet reproducibility guidelines.

Measurement of diffusing capacity can be helpful in assessing interstitial lung disease or emphysema. Some of these patients may have relatively normal spirometric findings. Recommended methods for performing diffusing capacity tests have been published and should be followed (204); reference equations are included.

Arterial blood gas measurement may be useful, particularly if there is some question about the degree of impairment indicated by spirometry. For patients with mild dysfunction or those with marked impairment by spirometry, arterial blood gas studies are not needed to assess impairment. The decision to obtain blood gas analysis should be made only after assessment of other examination data. One should remain mindful of the potential for associated morbidity and of technical factors that are important for obtaining a valid result. Equipment calibration, sample refrigeration, expedient analysis, breath holding or hyperventilation prior to the test, and the patient's position can all result in invalid measurements. Patients with significant interstitial disease may have a normal resting PaO_2, which becomes abnormal with exercise. Patients with marked airways obstruction may also have a normal resting PaO_2 that, in the absence of myocardial disease, may increase with exercise. Federal standards for PaO_2, adjusted for altitude, have been published (198). The significance of hypoxemia is often most obvious when it results in polycythemia, pulmonary hypertension, and cor pulmonale.

Maximum exercise testing is time-consuming and expensive but may be helpful in assessing the patient's ability to tolerate relatively brief high-energy demands. This test is often difficult for the patient and may be dangerous, especially for older patients. Furthermore, it is difficult to model the job energy demand. To avoid these problems, submaximal exercise may be used to estimate the maximum oxygen consumption per minute ($\dot{V}O_2$) from observation of heart rate and ($\dot{V}O_2$) (205). In the patient with both cardiovascular disease and lung impairment, these tests may be indicative of potential work capacity but do not define cause.

Clinical Care

Clinical management of coal miners with lung impairment is the same as for other patients with airways obstruction or interstitial disease. The care plan must be designed and adjusted individually with a goal of maintaining maximal function with minimal disability. The miner and his family must be educated about his disease and about how to treat it. Exertional dyspnea—the hallmark of pulmonary disability—can significantly reduce quality of life. The psychological effects of pulmonary disability on the patient and family should be explored and treated supportively. Sexual dysfunction may develop early and have devastating consequences. Any additional factors that lead to social isolation and diminished quality of life should be identified and treated where possible. For example, hearing loss may

be partially overcome through use of properly prescribed and fitted aids. Reduced strength resulting from chronic inactivity can be countered through graded exercise programs. Smoking miners must be directed to stop smoking and aided in the endeavor with appropriate support, referral, or pharmacologic measures. Techniques of "energy conservation" and "breathing retraining" help dyspneic patients avoid a sense of helplessness and loss of control. Pulmonary rehabilitation programs have been of significant benefit to patients with COPD and their families (206,207). Such programs utilizing multidisciplinary teams for education and treatment of miners and their families have been developed and supported in coal mining areas by the U.S. Department of Labor (208).

For miners with bronchitis and airways obstruction, good hydration and postural drainage together with bronchodilators and, if indicated, a trial with steroids often prove helpful. Early empiric antibiotic therapy may be helpful when there is evidence of pulmonary infection. Influenza and pneumococcal vaccines should be given at prescribed intervals. Continuous low-flow oxygen (1 to 3 L per minute) is indicated for patients with chronic hypoxemia (209,210). Sedatives and tranquilizers should be avoided, especially in patients with COPD. Congestive right-sided heart failure (cor pulmonale), a potential complication of advanced CWP, should be watched for and treated promptly. Finally, evidence of respiratory failure should be monitored closely.

Reduction of lung dust burden by whole-lung lavage is a therapeutic technique practiced in some areas of China, but it is used only as an exploratory technique in the United States. Although it appears to remove considerable amounts of deposited dust, cells, and other materials from the lungs, the long-term benefit of lavage to the miner has yet to be demonstrated (172).

Miners partially or totally disabled from their normal coal mine employment may be eligible for participation in state or federal benefits and compensation programs. Benefits may include limited or permanent income replacement as well as payment for medical expenses resulting from the pulmonary disability. The health care provider should facilitate referral to a knowledgeable counselor or agency able to inform the miner and his family about these programs.

PREVENTION

The means to prevent CWP and coal dust-related respiratory disease were provided in the Coal Mine Health and Safety Act of 1969 (5). These include primary prevention through dust control and secondary prevention through the use of medical screening. Research to improve dust controls and disease understanding was also mandated by the Act.

The Mine Safety and Health Administration of the U.S. Department of Labor is mandated to conduct regular mine inspections and monitor results from the operator dust sampling program. Since passage of the Act, marked reductions in average dust levels appear to have been achieved, well below the current standard (211–213). The exception to this is in highly productive mines using "longwall" methods, where dust control presents significant engineering challenges (214) and it has been difficult to attain mandated levels. Standardized methods for control of dust in the mine environment have been established (215).

The coal mine respirable dust standard of 2 mg per m^3 air was established to prevent the progression of simple CWP to PMF (216). A comprehensive review of the successes and limitations of this prevention strategy resulted in a new set of recommendations that NIOSH issued as a criteria document (217). This document noted progress in prevention but evidenced the persistent risk to miners of CWP and other pulmonary diseases. Updated recommendations were made to reduce dust exposure, improve hazard surveillance and exposure limit enforcement, and expand medical surveillance to include baseline and periodic tests of lung function as well as chest radiographs for all coal miners. Recommendations from NIOSH and other organizations were considered by the Advisory Panel on the Elimination of Pneumoconiosis empanelled by the Secretary of Labor (218). This committee issued the following findings and recommendations:

- Introduction of improved methods for inspection and enforcement of dust limits
- Enforcement of separate exposure limits for coal mine dust and silica dust that are lower than current limits
- Enhancement of hazard surveillance
- Expansion of health screening and surveillance to include lung function for all coal miners
- Improvement and expansion of training in dust control

Although great progress has been made in reducing occupational disease in coal miners (e.g., Fig. 22.1), concerns are periodically raised about adequacies of dust control and whether the dust samples analyzed by the Mine Safety and Health Administration accurately reflect dust conditions in the mines (219–221). Hence, the risk of disease remains uncertain, and sustained preventive interventions are critical. New dust assessment and control initiatives, such as the machine-mounted continuous respirable dust monitor, may assist in this aim.

Working miners with evidence of CWP, as identified under the National Coal Workers' Surveillance Program administered by NIOSH, are entitled to work in a low-dust area within the mine (currently less than

1 mg per m^3 air) and to have their exposures monitored more frequently (222). In this program, miners receive a mandatory chest radiograph on entry into the work force and after 3 years. Thereafter, voluntary radiography is offered every 5 years. Films are classified by trained readers, and the results are sent to the miner and a personal physician if one is designated, but not to the employer. If evidence of radiographic CWP is found at the category 1 level or higher, the miner is informed that he or she is eligible for job transfer. If the miner opts to transfer to a low-dust area of the mine, there is no immediate loss of pay for the miner. Miners should be encouraged to participate in the surveillance program and exercise the transfer option when eligible.

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