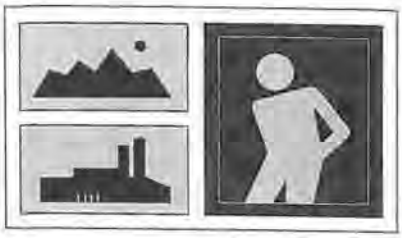
Environmental and Occupational Medicine, Third Edition, edited by William N. Rom. Published by Lippincott-Raven Publishers, Philadelphia, 1998.



CHAPTER 28

Respiratory Disease in Coal Miners

Michael D. Attfield and Gregory R. Wagner

Coal miners develop a variety of lung diseases as a result of their workplace exposures. Of these, coal worker's pneumoconiosis (CWP) has received the most attention, perhaps because of its clear occupational association. Bronchitis and emphysema resulting from coal mine dust exposure, clinically indistinguishable from their nonoccupational analogues, are more 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 United States 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 around 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 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/m^3 air, later reduced to 2 mg/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 right of entry for research to advance understanding of the health effects of mining.

Coal worker's pneumoconiosis is a well-defined medical entity resulting from the deposition of coal mine dust in the lung and from 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 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.

The United States has extensive coal deposits (Fig. 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

M. D. Attfield: Epidemiology Investigations Branch, National Institute for Occupational Safety and Health, Morgantown, West Virginia 26505-2888.

G. R. Wagner: Division of Respiratory Disease Studies, National Institute for Occupational Safety and Health, Morgantown, West Virginia 26505-2888.

FIG. 1. Coal deposits in the United States 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. (Adapted from ref. 211.)

mine dust exposure and the development of respiratory diseases in order to diagnose, treat, and prevent them.

EPIDEMIOLOGY

Perhaps more investigation has been directed toward lung diseases of coal miners than toward any other occupational disease. Meiklejohn (8–10), for example, cites more than 100 reports and articles published before 1950 on the health of coal miners. 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. More recently, 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 around the world. In addition, these intensive multifaceted studies have clarified many aspects of the causation and significance of lung disease in coal miners.

General Morbidity

Recognition that coal miners were at risk of lung disease due to their work came late to the United States. As a consequence, active epidemiologic investigation of coal miners did not start until after 1960. Between 1960 and 1970, however, seven studies were undertaken in various regions of the country (12–18). The findings from these showed that a risk of CWP existed in all regions studied, but that it varied considerably, depending on rank of coal (Fig. 2). (Coal rank is associated with the degree of metamorphosis of the coal due to heat and pressure and is often measured by the percentage of carbon. Anthracite has the highest percentage of carbon and gives rise to the most severe disease.)

A significant restriction on respirable dust levels in U.S. coal mines was mandated by the 1969 Federal Coal Mine Health and Safety Act (5). CWP prevalence rates from 1970 to the present are shown in Table 1. These figures were derived from a large, long-term, nationwide study of lung diseases of coal miners in the United States (19) and standardized to a common tenure distribution obtained from a survey of coal mine employment in 1986 (20). Standardized rates derived from data collected by the federally operated Coal Worker's X-Ray Surveillance Program (CWXSP) (21) are also shown in the table. Both sets of information indicate a declining trend in prevalence. The observed trends may not be due solely to the lower dust levels, as miner participation in these programs has been suboptimal.

Coal miners tend to report more respiratory symptoms and have poorer lung function than control groups (17,22–25). In some cases a parallel effect has also been seen when miners' wives were compared to wives of the nonminer control group, suggesting that environmental

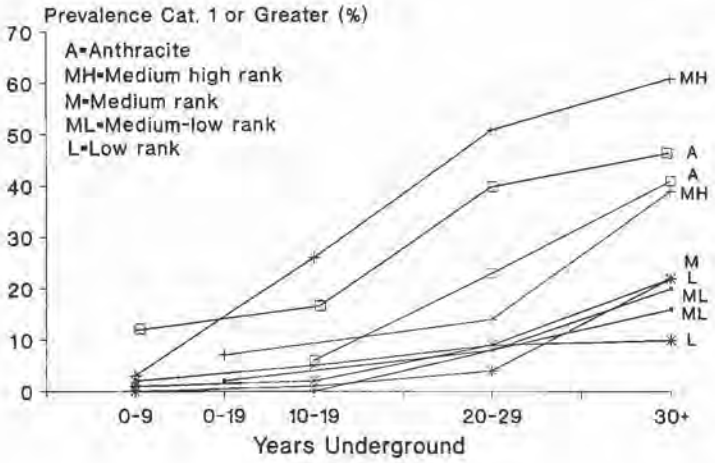


FIG. 2. Prevalence of category 1 or greater small rounded opacities against tenure in mining by coal rank group for seven studies (one including three regions) undertaken before 1970 in the United States.

and social factors may also be involved (22,24). Higgins (23) reviewed findings from both the United States and Britain and concluded that there was consistent evidence of greater prevalence of respiratory symptoms and lower average lung function in miners and ex-miners than in nonminers.

General Mortality

Mortality patterns among coal miners have been studied systematically for about 100 years, beginning with British occupational mortality statistics for 1890 to 1892. These studies have generally shown increased standard mortality rates (SMRs) for accidents, respiratory disease, respiratory tuberculosis, and stomach cancer. Overall, SMRs from all causes have varied somewhat, from a high of 195 in an early study (26) to levels slightly above 100 in a number of more recent large analyses (27–30). The later studies reveal evidence of healthy worker selection effects, some reports showing statistically significant elevations in SMR for ex-miners and for current miners who likely moved to surface jobs because of ill health.

Standardized rates for death from pneumoconiosis have consistently shown increased risks associated with progressive massive fibrosis (PMF) (29–36). Most of the studies, however, failed to detect any elevation in mortality risk associated with the presence of simple CWP. Miller and Jacobsen (30) found that simple CWP was associated with a 2% to 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 (age 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 (27) found increased rates of emphysema, influenza, asthma, and tuberculosis, whereas Miller and Jacobsen (30) 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 was recently reported for nonmalignant respiratory disease among Dutch coal miners (37).

Two consistent features of coal miner death rates relate to cancers of the lung and stomach. In the former case the standardized rates typically have been low (38), and no obvious relationship of lung cancer mortality with dust exposure has been detected (39). An excess of deaths due

TABLE 1. *Rate of category 1/0 or greater, and 2/1 or greater small rounded opacities standardized to a common tenure distribution representing the work force distribution in 1986 for the NSCWP and CWXSP*

		Adjusted summary prevalence			
	Category	1970–72	1973–75	1977–81	1985–88
Epidemiologic data (NSCWP)					
Common tenure	1/0+	6.6	5.1	3.6	2.3
Distribution (%)	2/1+	1.5	1.2	0.5	0.3
Surveillance data (CWXSP)					
Common tenure	1/0+	10.7	9.9	7.3	3.6
Distribution (%)	2/1+	2.4	1.2	0.7	0.4

NSCWP, National Study of Coal Workers' Pneumoconioses; CWXSP, Coal Worker's X-Ray Surveillance Program.
Note: Rates from the CWXSP are for the second readers (all B readers).

to stomach cancer has been seen among miners in Britain (28,40), the United States (27,41), Holland (37,42), and Japan (43). There is a suggestion that stomach cancer risk is related to exposure to coal mine dust (30). Ames (44) describes a number of hypotheses that could explain the higher gastric cancer mortality in coal miners. Among these is Meyer et al.'s (45) hypothesis that stomach cancer incidence would be greater in miners with good lung clearance, since the dust would be transported from the lungs and then swallowed. Some findings of Dutch coal miners support this view (42), although it was not found for U.S. coal miners (39).

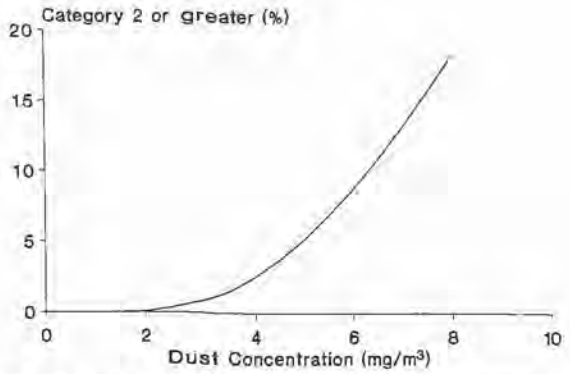


FIG. 3. British miners' predicted risk of contracting category 2 or greater small rounded opacities over 35 years plotted against dust concentration.

Morbidity, Coal Mine Dust Exposure, and Other Risk Factors

Coal Worker's Pneumoconiosis

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 (46–50). Coal rank 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 (46,47). However, quartz was implicated in a study of a group of cases of unusually rapid progression of simple pneumoconiosis (51). In addition, miners with a particular form of PMF that appeared to consist of conglomerations of the larger nodules of simple CWP (type *r* (123) opacities) had received higher exposures to quartz than had their controls (52).

Among nonoccupational factors, smoking was not found to affect simple CWP development (53), nor did bronchitis appear to play a role (54). However, an important risk factor for the development of PMF is presence of simple CWP (49,55,56), and risk increases with category of disease. There are also indications that body mass and breathlessness may be positively related to future development of PMF (57). [The latter result agrees with pathologic findings that show widespread emphysema in miners with PMF (58).] 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 at all in the remaining three (59).

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 (46). Figure 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 (55), 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 progressive massive fibrosis 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/m^3, that dust concentration was adopted as the federal standard.

Recent British analyses of exposure and response have concentrated on PMF as the response variable(49) (Fig. 4). These findings were derived from study of 52,264 5-year intervals of risk for more than 30,000 British miners. Probabilities of contracting simple CWP or PMF, or of progressing from one category of simple CWP to another, were modeled against initial category, dust exposure, coal rank, and worker's age. The results were used to obtain predicted probabilities for 5-year periods, and these predictions were then compounded into 40-year risks. Clear

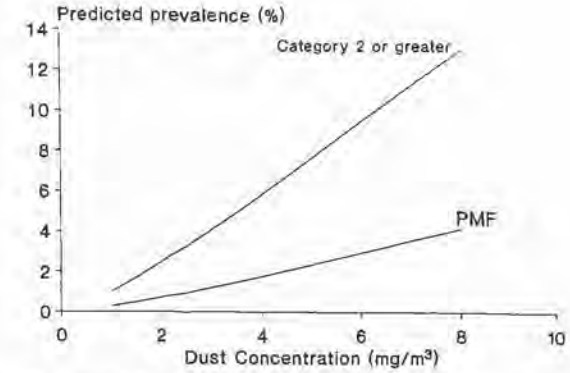


FIG. 4. British miners' predicted risks of contracting category 2 or greater small rounded opacities and PMF after 40 years work plotted against dust concentration.

exposure-response relationships were found for dust and coal rank, the predictions of risk being somewhat greater than those from earlier 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 appeared to be dependent on the degree of previous dust exposure. This implies that PMF could not be eliminated exclusively through control of simple CWP.

Exposure-response relationships have been reported among miners from countries other than Britain. Reisner (60,61) 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/m^3, and less than 4% for 2 mg/m^3 (62).

In the United States, data collected in the last few years, before federal regulation caused a reduction in dust levels, were used to compute retrospective dust exposures for miners working at round 1 of the National Study of Coal Workers' Pneumoconiosis (NSCWP) (63) and then applied to estimation of exposure-response for CWP (64). 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 Fig. 5, which provides predicted prevalences of category 1 or greater, 2 or greater, and PMF for a range of dust exposures. These predictions tend to be somewhat higher than those derived from studies of British and German miners (49,62). These findings were confirmed in another study involving U.S. underground coal miners and ex-miners medically examined between 1985 and 1988 (65).

Most of the studies noted above were undertaken on working miners and were therefore vulnerable to bias induced by ignoring miners in poor health who left mining. Such a bias was apparent in older, higher dust exposed miners (66), while Attfield and Seixas (65) 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, "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" (67). A similar observation was made regarding development of PMF in current and ex-miners (68). (These observations should be contrasted with those for ventilatory function discussed below, where there appears to be evidence of more extreme effects of dust on those who leave 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 (60) support the concept of a "residence time" effect, an observation confirmed to some extent by Hurley and colleagues (48). In an exploration of modeling approaches, Attfield et al. (66) 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. Category of simple CWP remains a strong predictor of PMF development, but occurrence of PMF has been found to be related to dust in the absence of simple CWP. Recently developed exposure-response relationships indicate that CWP incidence for dust levels of 2 mg/m^3 air or less may be greater than was predicted in the past.

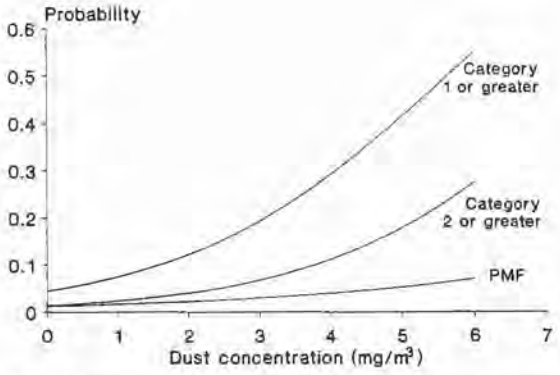


FIG. 5. U.S. miners' predicted risk of contracting category 1 or greater small rounded opacities, 2 or greater small rounded opacities, or PMF after 40 years work in coal mining.

Small Irregular Opacities

Epidemiologic researchers in the United States have tended to define simple CWP in terms of the so-called combined opacity-profusion determinations. That is, they took into account both rounded and irregular types of opacity. British studies, in contrast, have generally treated simple pneumoconiosis as being synonymous with small rounded opacities (69). Recently, however, there has been increasing interest in the relationship of irregular opacities per se to both dust exposure and to symptoms and lung function.

As with small rounded opacities, the prevalence of small irregular opacities increases with occupational dust exposure and is linked to reduced lung function (58,70,71). Elevated respiratory symptom levels have also been observed in miners who have irregular opaci-

ties (71), but epidemiologic study of the medical implications of the type of opacity (irregular versus rounded) is confounded by reader disagreement on opacity type. The latest information on British miners indicates little difference between the prognostic implications of the two types of opacity for future ill health (69), so opacity profusion rather than opacity type should remain the primary radiographic variable of interest.

Other Lung Diseases

Correlation of measured dust exposures with indicators of lung disease other than pneumoconiosis have 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 (72). Findings for United States, Australian, German, and Sardinian coal miners support these observations (73–76).

Cross-sectional studies of 1-minute forced expiratory volume (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 (67,73,77–83). 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 presence of CWP on FEV_1 was noted. Some suggestion of a greater dust exposure effect among ex-miners has been reported, but this observation could have been due to chance (67). 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. This finding, some have suggested, 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 (78), 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 (84). Logistic models fitted to responses based on reports of persistent cough and phlegm, FEV_1 less than 80% of predicted value, reduced FEV_1 and cough and phlegm, and FEV_1 less than 65% of predicted all showed significant relationships to dust exposure. The prevalence of the four responses at high dust exposures was found to be close to that among smokers who had hypothetically zero dust exposure (Fig. 6).

Longitudinal changes in ventilatory function in coal miners have also been linked with dust exposure (69,76,85–89). However, the relationship is complex, and varies depending on the age (or, more likely, prior mining tenure) of the miners (89). New miners appear to suffer a fairly severe initial decline after beginning work in mining (89–91). This loss is then ameliorated, but it is still detectable in experienced miners (85,86). In a study of young miners by Carta and colleagues (76), 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 selection, whereby those able to withstand the higher dust exposures remain in the dusty jobs. Support for this is found in the study by Petsonk and colleagues (92), which showed that miners with greater airway responsiveness were less likely to work in dusty jobs.

The general effect of dust exposure on FEV_1 seems to lie around 0.7 ml of FEV_1 per gram-hours/m^3 (about 5 ml per year for a dust exposure of 4 mg/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 being observed in a relatively healthy population fit enough to work in an arduous job. If a close look is taken at 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., about 5 ml per year per pack smoked (19). 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.

Comparison of average effects of smoking and dust exposure has been said to paint a misleading picture of

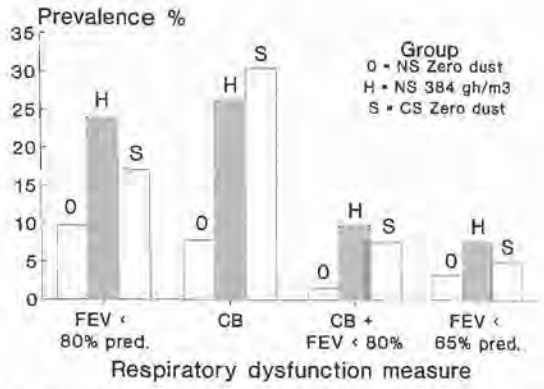


FIG. 6. Predicted prevalence of four indices of respiratory dysfunction for never smokers at 0 and 384 gh/m^3 (gram-homs/cubed meter) air and current smokers at 0 gh/m^3.

their true effects (93). It has been argued that the similar average effects seen in the various studies involving regression modeling of FEV_1 (78) arose through different mechanisms, the average decrement associated with smoking being due to a subset of severely affected smokers, and the dust exposure effect arising from a small general shift in all exposed miners. If this argument were true, it would imply that dust exposure is of trivial significance, and that its effect should not be equated with that arising from exposure to tobacco smoke.

There is little evidence to support this conjecture. First, no sign of excess numbers of severely affected smokers (compared to those who never smoked) was seen in an analysis that looked at the distribution of FEV_1 values for a group of working U.S. coal miners for whom a definite dust exposure effect had been previously established (65). Rather, the results indicated that the miners were reacting similarly (in terms of FEV_1) to exposure to tobacco smoke and to dust. Second, a study of British miners identified a group of ex-miners who apparently had suffered a severe response to their dust exposure (67,94). The authors concluded that this indicates that dust exposure can give rise to severe respiratory impairment in the absence of PMF. Lastly, in an analysis of autopsy material on coal miners, Leigh and coworkers (95) found a clear inverse relationship between extent of emphysema and FEV_1 in both smokers and nonsmokers, while emphysema and lung dust content were positively related. These results suggest a dust-related loss in FEV_1 not of trivial significance.

In contrast to the study of exposure-response for CWP, where extensive exploration of various dust composition parameters (e.g., coal rank, 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 to the contrary, an understanding confirmed by the results of Coggon and colleagues (96). These show wide variations in the 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.

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 coal miners. In this respect, one case report exists concerning a miner reported to have occupational asthma due to *Rhizopus nigricans* (97). Mold colonies were found extensively in the mine in which the case worked, including *Rhizopus nigricans* and other genera.

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.

Mortality, Coal Mine Dust Exposure, and Other Risk Factors

In two studies looking at mortality in association with quantitative measures of cumulative dust exposure (30,98), mortality from pneumoconiosis increased with extent of dust exposure. Additionally, and importantly, mortality from bronchitis and emphysema was also related to dust exposure in both studies (p <0.001 and <0.01, respectively). The relationship with bronchitis and emphysema implies that dust exposure per se, even in the absence of radiographic abnormality, gives rise to excess mortality.

Surface Coal Miners

Compared to the sizable number of studies and reports on underground coal miners, surface miners have been 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 so 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 (99,100). In an initial study (99), 4% of the workers showed signs of CWP but only 7 of 1,438 had 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 subsequent reports of acute silicosis among surface miners working in drilling operations (101). 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. 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 pathologically diagnosable early signs (25% sensitivity when macules only are recorded; 40% for macules or mild degrees of micronodules), although its specificity appears to be good [90% specific for ten films with no observed pathologic abnormalities related to CWP (102,103)]. Other dust diseases of miners are usually undetectable by plain chest radiography (104). This discussion of radiography, therefore, 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. Fibrosis is usually noted first in the upper or middle lung zones and predominates there, but potentially it is visible anywhere. In simple CWP, opacities vary in size from 1 mm to 1 cm. By radiographic convention nodular opacities 1 cm and larger are defined as PMF.

Smoking and aging are thought by some to influence the profusion of irregular opacities (105), although in the absence of dust exposure interstitial opacities are rarely found in healthy workers (106,107). A radiograph with predominantly irregular opacities in a coal miner raises the possibility of previous exposure to occupational hazards such as asbestos or talc or of a nonoccupational interstitial lung disease. The large opacities of PMF almost invariably occur on a background of simple CWP. They are usually rounded, may be multiple in number, and are most often found posteriorly in the upper lung zones. PMF may affect an entire lobe (Fig. 7).

The differential diagnosis of the small opacities of simple pneumoconiosis includes (1) diseases that produce acute nodular lesions, such as miliary tuberculosis and viral pneumonia; and (2) diseases that produce chronic nodular patterns such as other pneumoconioses, metastatic disease, tuberculosis, and other granulomas. Silicosis presents with the same radiographic pattern as CWP and so can be differentiated only by occupational history or pathologic examination. The large opacities of PMF must be differentiated from malignancies, granulomatous diseases, and other less common causes.

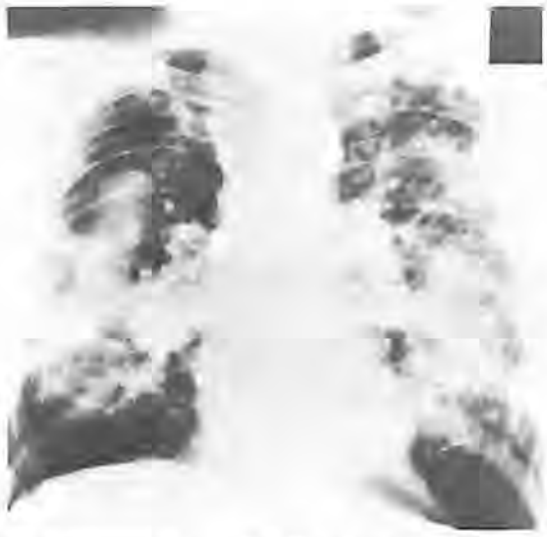


FIG. 7. Progressive massive fibrosis, category C, of coal worker's pneumoconiosis.

To improve recognition and reporting of CWP and other pneumoconioses, international standards have been adopted. These classification methods have contributed materially to epidemiologic studies of coal miners, to medical surveillance programs, and to 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 as B readers. Their interpretations are generally more consistent with one another and are given more weight in both epidemiologic investigations and legal proceedings. Nevertheless, there remains significant inter- and intrareader variability, particularly with mild disease (108–110).

More technologically advanced imaging methods such as high-resolution computed tomography (HRCT) have been promoted by some for the diagnosis of coal worker's lung diseases (111,112). To date there is no standardization of interpretation of the changes identified on CT, nor has epidemiologic investigation been sufficient to establish relationships between levels of exposure to coal mine dust and CT abnormalities (113,114). For now, CT should be utilized when there are specific clinical indications or when it is part of a research protocol; routine use for surveillance of miners is not indicated. Use of gallium-67 citrate imaging should be similarly limited (115).

PATHOLOGIC FEATURES

The descriptive work of Heppleston (116) and Gough and coworkers (117) 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 macule, occurs specifically among workers exposed to coal mine dust, regardless of geographic location, rank of coal, or type of dust. The 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" (118). 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.

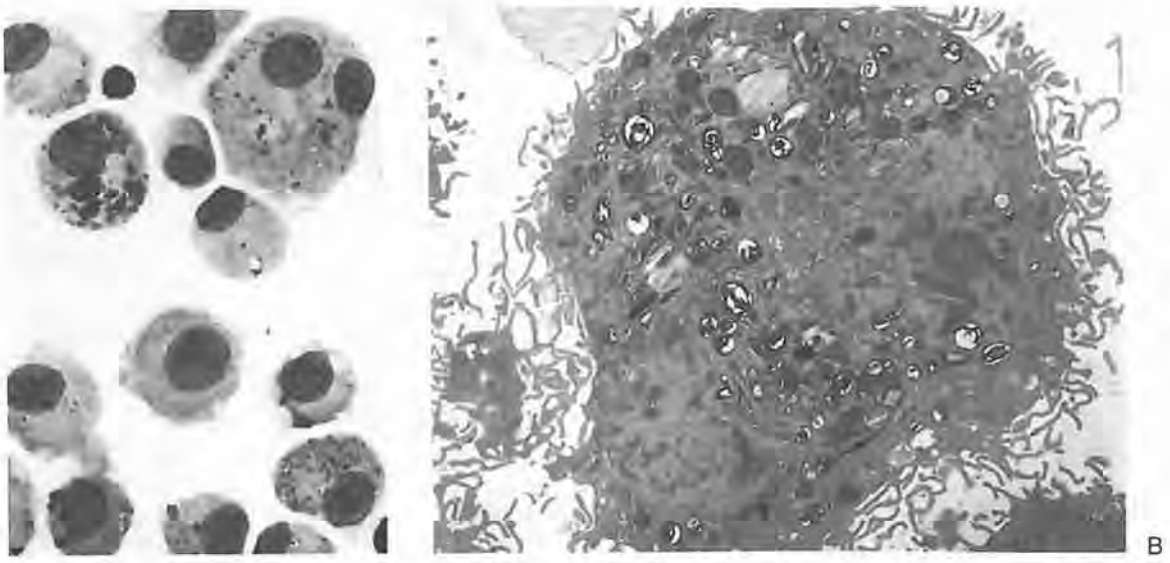


FIG. 8. Coal-laden macrophages recovered by bronchoalveolar lavage (BAL) from an active coal miner. **A:** Variably shaped dark particles in light microscopic study of alveolar macrophages (×400). **B:** TEM study of alveolar macrophage from a coal miner shows particles in phagolysosomes (×4,200). (Courtesy of T. Takemura, M.D., and V. Ferrans, M.D.)

FIG. 9. The coal macule surrounding a dilated respiratory bronchiole.

Macrophages laden with coal mine dust are found free in the alveoli of anyone who inhales coal mine dust; they have no specific pathologic meaning (Fig. 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- to 2-mm zone of enlarged air space referred to as focal emphysema (Fig. 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 (119–121). Bituminous and anthracite coal can usually be distinguished by light microscopy (122).

The nodular lesions of CWP have been classified as micronodules (up to 7 mm diameter) and macronodules (7 to 20 mm diameter) (118). 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. 10). Degenerative changes, including calcification, cholesterol crystallization, blood vessel obliteration, and infarction, are commonly observed.

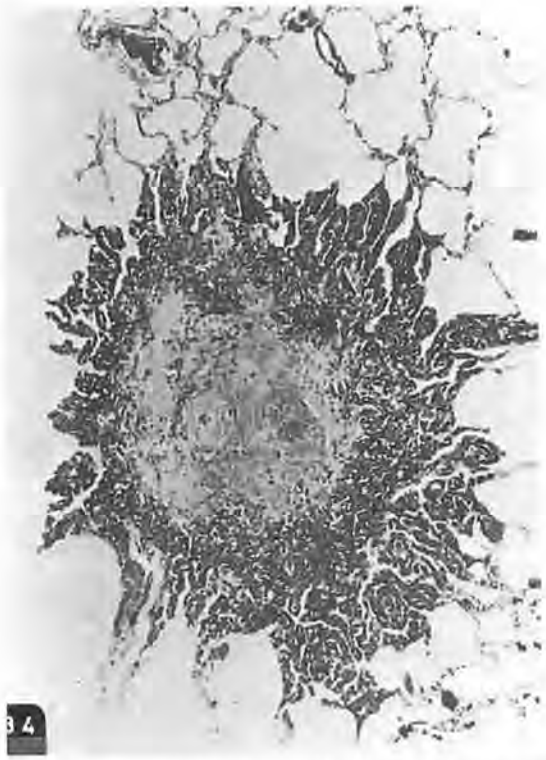


FIG. 10. A macronodular lesion of coal worker's pneumoconiosis has a center that is becoming hyalinized.

FIG. 11. This whole lung section shows progressive massive fibrosis of coal worker's pneumoconiosis against a background of nodules, macules, and focal emphysema.

The pathologic definition of PMF is arbitrarily based on the diameter of lesions. Fibrotic pneumoconiotic lesions greater than 1 cm have been accepted as radiographic evidence of PMF. This definition was adopted in the regulations of the National Coal Workers' Autopsy Study mandated by the 1969 Federal Coal Mine Health and Safety Act. However, the pneumoconiosis committee of the College of American Pathologists recommended a minimum diameter of 2 cm for morphologic investigations (118) despite the radiographic definition of PMF as nodules 1 cm or greater in diameter (123).

The 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 destroy blood vessels and airways and greatly distort lung architecture (Fig. 11). The larger PMF lesions tend to cavitate, sometimes discharging a black liquid into a communicating airway. 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 of the lesions, and an obliterative vasculitis is observed in peripheral areas (124).

Rheumatoid pneumoconiosis (Caplan's syndrome) is characterized by large (1 to 5 cm) 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 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. 12). A study of 3,365 U.S. underground coal miners' autopsies revealed that about 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 (125).

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 (126–128). Weight for weight, the mineral (noncoal) portion of the dust appeared to be more responsible for the radiographic opacities than the coal fraction. This implies that miners in high-rank coal mines would have to retain appreciably

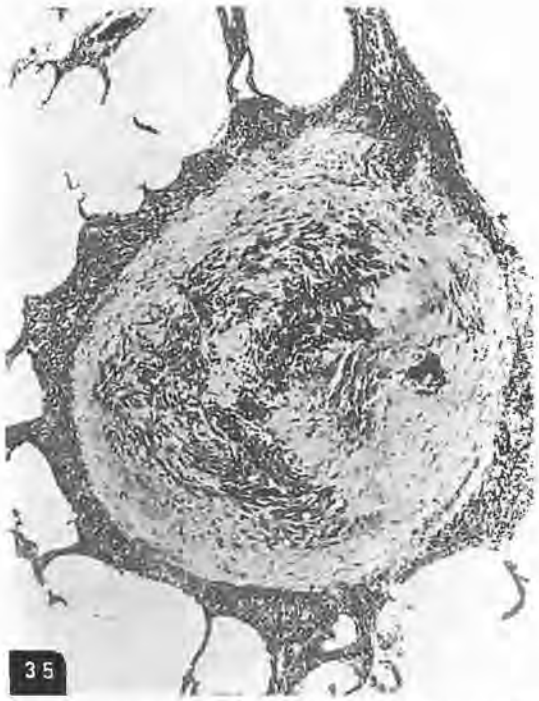


FIG. 12. Silicosis nodule with paracicatricial emphysema in a coal worker's lung.

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 (127), it was suggested (128) that radiographic changes are related simply to dust accumulation.

Additional work has supported the link between amount and composition of retained dust and opacity profusion on radiographs (129). It also revealed that the relationships between radiographic data, lung content, and lung disease are rather more complex than was originally supposed. In this respect, it is clear that the size (radiographic type) of the opacity must be taken into consideration. In both high- and low-coal rank areas, miners with the smallest type of rounded opacities (type *p*) had greater lung dust weights than miners with type *r* opacities, after the data was controlled for degree of profusion (129). 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 radiopaque. 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* or *r* (130). For the largest type (*r*) and a high proportion of ash in the retained dust, no association between profusion and dust weight was found (130), 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 (130)]. 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), though nodules of >3 to 9 mm 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.

All in all, 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 on the radiographs 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 (52) and pathologic data (131). A study undertaken to compare radiographic and pathologic appearances of PMF provided some support for this hypothesis (132).

There is a clear correlation between dust exposures and retained dust in the lung (133,134), between retained dust and presence and severity of pathologic lesion (133,134), and between dust exposure and presence and severity of pathologic lesion (134). Pathologic abnormality was better predicted by retained dust than using dust exposure (134). Miners with PMF appeared to have retained more dust per unit of exposure than those without PMF (133). 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, low-rank coal miners had higher levels of ash in the retained dust, the ash content being greater proportionately than it was in the mines (133). 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 (135–137). 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 (58,138,139). Not only was the excess of emphysema among coal workers confirmed in a study that controlled well for bias (58), but

emphysema was also found to be related to both dust retained in the lungs (58,129) and to pathologic measurements of pneumoconiosis (129). The finding that miners with PMF also have elevated amounts of emphysema unrelated to the PMF lesions(58) is consistent with the epidemiologic finding that dyspneic miners were at greater risk of PMF development (57). Importantly, the presence of emphysema was related to dust exposure during life (129), particularly the coal rather than the silica component (139), and to FEV_1 percent predicted (95,139). These findings therefore indicate a causal relationship between dust exposure and emphysema, and the potential for ensuing disability. There is some indication that irregular opacities on chest radiographs are associated with pathologic signs of emphysema and interstitial fibrosis (58).

Despite the clear association between occupational exposure and chest symptoms reported by Rae and coworkers (72), results from the few pathologic studies of bronchitis have been mixed. Leigh and colleagues found no evidence of an association between bronchial mucous gland-wall ratio and years worked underground (139), even though other studies of Australian miners had shown correlations between gland-wall ratio and various symptoms and signs of bronchitis (140,141). However, a study using measured dust exposures rather than the surrogate years underground (142) found that maximum mucous gland-wall ratio did correlate with lifetime occupational exposure. Douglas and colleagues (142) said their results "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

Elucidation of the mechanisms involved in the development and progression of coal mine dust–induced pulmonary disease has been a topic of extensive investigation. Most theories on the cause of mineral dust–induced lung injury involve the role of free radicals and oxidative damage (143). It has been shown *in vivo* that exposure of rats to mineral dust enhanced the release of reactive oxygen species from pulmonary phagocytes and was associated with oxidant damage to the lung parenchyma (144,145). Indeed, treatment *in vivo* of animals with scavengers of superoxide, hydrogen peroxide, and hydroxyl radical was effective in decreasing lipid peroxidation and edema associated with pulmonary inflammation (146). In addition, superoxide secretion from alveolar macrophages has been shown to decrease α_1-antiprotease activity, which may represent an alternative mechanism that renders the lung subject to elastase damage and emphysema (147). Inhalation of coal dust has also been shown to increase the production of the reactive nitrogen species (nitric oxide) and to enhance messenger RNA (mRNA) levels for the inducible form of the nitric oxide synthase enzyme (148).

Studies *in vitro* indicate that coal dust was lytic and resulted in the release of lysosomal enzymes from alveolar macrophages (149). In addition, *in vivo* exposure of alveolar macrophages to coal dust resulted in release of inflammatory mediators that have been characterized by abnormal gallium lung scans and clearance of inhaled technetium diethylenetriamine pentaacetic acid (DFTPA) aerosol (150). In general, studies *in vitro* suggest that coal dust is a less potent stimulant than silica. Such a relationship correlates with the pathogenicity of these dusts *in vivo*.

Lebeder and co-workers (151) have reported that crushing coal resulted in the generation of free radicals on the cleavage planes. The presence of these coal radicals has been associated with the lytic potential of the dust (152). Freshly fractured, but not aged, coal dust has been shown in vitro to increase the release of proinflammatory metabolites of arachidonic acid, such as prostaglandin E_2 (PGE_2) and thromboxane A_2 (TXA_2) (153). A relationship has also been found between the pathogenicity of anthracite versus bituminous coal and the reactivity of radicals generated after grinding coal of different ranks (152). Pathologic studies of coal miners' lungs indicate that lungs with PMF contained higher levels of coal free radicals than less severely diseased lungs (152). This may be due in part to higher levels of retained dust in lungs with more extensive disease.

Animal studies also support the theory that exposure to coal dust results in excess oxidant load in the lungs. Inhalation exposure of rats to coal dust (2 mg/m^3, 7 hours per day, 5 days a week, for 2 years) resulted in the activation of alveolar macrophages. Activation was expressed as an increase in surface ruffling and cell spreading and enhancement of the spontaneous and particle-stimulated levels of chemiluminescence (an indicator of production of reactive species) (154). Animal studies also indicate that inhalation of coal dust increases the number of alveolar macrophages (154–156) and neutrophils (157) obtained by bronchoalveolar lavage. Elevated levels of blood and interstitial monocytes have also been noted following animal exposure to coal dust (158). This recruitment of phagocytes may be due to the coal dust-induced release of leutotriene B_4 from alveolar macrophages (159).

Such activation of alveolar macrophages has also been reported in coal miners with pneumoconiosis. Wallaert and colleagues (160) reported that bronchoalveolar lavage yielded more alveolar macrophages from coal miners with pneumoconiosis than from controls. In addition, chemiluminescence was elevated in miners with measurable pulmonary disease. The relationship between CWP and activity of alveolar macrophages was also reported by Rom and co-workers (161,162). Their results indicate that CWP was associated with enhanced secre-

tion of superoxide anion and hydrogen peroxide from alveolar macrophages. Elevated levels of elastase complexed with α_1-antitrypsin and increased secretion of fibroblast proliferative factors (fibronectin and alveolar macrophage-derived growth factor) were also noted (161,162). Morphologic studies also support the hypothesis that alveolar macrophages are activated in CWP (163). Cells contained more particles; exhibited more ruffling, filopodia, and phagolysosomes; and demonstrated more cell fusion and mitosis. It is interesting that the number of coal dust particles within each phagocytic cell was not different for currently exposed miners and those who had not been exposed for at least a year (163). This suggests that lung burden in lifelong coal miners can remain high long after occupational exposure has ceased.

There is evidence that the magnitude of activation of alveolar macrophages in coal miners is directly related to the extent of pulmonary injury. Wallaert and coworkers (160) reported that macrophages from coal miners with progressive massive fibrosis generated more chemiluminescence than macrophages from miners with simple pneumoconiosis. Rom (164) reported that macrophages harvested from individuals with mineral dust exposure but no impairment secreted significantly lower levels of fibronectin, alveolar macrophage-derived growth factor, superoxide, and hydrogen peroxide than those with radiographic and spirometric evidence of disease. Lapp and colleagues (165) reported that although asymptomatic coal miners did not exhibit enhanced chemiluminescence, their alveolar macrophages were significantly more ruffled, suggesting some level of activation.

Recent studies with coal miners have concerned the identification of cytokines and growth factors, which correlate with disease severity. Lapp et al. (166) reported that bronchoalveolar lavage fluid levels of tumor necrosis factor (TNF-α) are decreased in coal miners without CWP. In contrast, miners with CWP exhibited elevated release of TNF-α from alveolar macrophages and blood monocytes (167,168). In addition, mRNA levels for TNF-α are elevated in alveolar macrophages of miners with CWP (169). TNF-α is an initiating cytokine that has been correlated with the release of chemoattractants and fibroblast growth factors (170,171). Therefore, it is not surprising that antibodies to TNF-α have been shown to suppress fibrosis in animal models (172) and that TNF-α secretion from alveolar macrophages correlates with disease severity in coal miners.

The production of fibrogenic factors by alveolar macrophages has also been reported for coal miners with pneumoconiosis. Both type I insulin-like growth factor (a progression factor) and platelet-derived growth factor (a competence factor) are elevated in miners with CWP and to a greater extent in coal workers with PMF (173). In contrast, interleukin-6, a cytokine that may be antifibrogenic (174), is elevated in asymptomatic miners (166) and tends to decrease with increasing disease severity (169). These data suggest that the balance between protective and damaging cytokines controls the initiation and progression of disease.

PULMONARY PHYSIOLOGY

Cross-sectional and longitudinal epidemiologic investigations, reviewed above, have provided most of the information currently 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 a relatively high prevalence of dyspnea, chronic bronchitis, and chronic obstructive pulmonary disease in addition to CWP. Pathology investigations demonstrate elevated levels of emphysema in miners (175). Coal mine dust exposure and cigarette smoking have both been found to be unambiguous risk factors in 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 those without CWP (78,176). These studies attempt to correlate two different effects of coal mine dust exposure: development of CWP and 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 (15,177). Nevertheless, a matched sample analysis of a large number of American 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 (178). 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 category B and C of that disorder (15,177, 179,180). Coal mine dust exposure per se has been associated with accelerated loss of FEV_1 in longitudinal studies and reduced FEV_1 in both cross-sectional field studies and in laboratory investigations (see other lung diseases in Epidemiology, above).

Studies of lung volume in coal miners have revealed a slight increase in total lung capacity (TLC) among obstructed and nonobstructed miners (181). Those without airways obstruction have been found to have consistent increases in residual volume (RV), which tended to increase with the radiographic category of CWP. Similarly, studies of dynamic compliance, thought 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 (176). The clinical significance of these early physiologic changes is not yet clear.

Diffusing capacity has been found to be reduced among those with predominately type *p* opacities of simple CWP, among these with category B and C of PMF, and among smoking miners. Nonsmoking miners have generally been found to have diffusing capacity measurements within the normal range (182–184)

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 airway obstruction tend to show lower PaO_2 during exercise than those without obstruction (185). Significant decreases in PaO_2 with exercise have been found among miners with PMF (186). Nonsmoking miners have been found to have a lower PaO_2 and higher alveoloarterial oxygen difference than nonsmoking nonminers, both at rest and with exercise (83).

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 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, 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 normal coal mine job.

A history of chest infections or chest trauma may be important. Medical records, especially previous chest radiographs and lung function tests, are often helpful to the medical assessment. Harmful respiratory tract exposures (e.g., asbestos, silica) in the home or in prior workplaces may be important. 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) reason for stopping. A subjective measure of dyspnea, cough, and phlegm is useful. Questioning what kinds of 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's Epidemiologic Standardization Project Questionnaire (187), which also contains questions aimed at assessing cough, phlegm, and wheezing. It 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 (for example, 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 look broadly at the health of the miner and to 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 histories 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 Organization (ILO) classification to assess pneumoconiosis, while paying particular attention to other thoracic abnormalities. The electrocardiogram (ECG) 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 (188). The FEV_1 is the single most useful measurement of the spirogram. It is reasonably reproducible, less effort dependent than the FVC or maximal voluntary ventilation (MVV), 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 dependent on a full and reproducible FVC. The MVV 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 by 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 (189–191). Also worth considering is that working people as a rule are healthier than the average person when they begin employment (192). 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 (193). Recent analyses have highlighted the fact that impaired workers may have more variable spirometric findings than healthier ones (194). 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 test have been published and should be followed (187). Reliable prediction equations are available (195).

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, 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 airway 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 (188). 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 (VO_2) from observation of heart rate and VO_2 (196). In the patient with both cardiovascular disease and lung impairment, these tests may be indicative of potential work capacity but they 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 (197,198). 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.

For miners with bronchitis and airway 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 antipneumococcal vaccines should be given at prescribed intervals. Continuous low-flow oxygen (1 to 3 L per minute) is indicated for patients with chronic hypoxemia (199). 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 routinely practiced in China but only as yet attempted as an exploratory technique in the United States. Though it appears to remove considerable amounts of deposited dust, cells, and other materials from the lungs, its long-term benefit to the miner has yet to be demonstrated (200).

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.

The Mine Safety and Health Administration of the 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 (Fig. 13) (201–203). The exception to this is in the highly productive growing number of long-wall mines, where dust control presents significant engineering challenges (204), and it has been difficult to attain mandated levels.

The dust standard of 2 mg/m^3 air was established to prevent the progression of simple CWP to PMF (205). A recent comprehensive review of the successes and limitations of this prevention strategy resulted in a new set of recommendations for NIOSH issued as a criteria document (206). This document noted progress in prevention but 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, expand medical surveillance to include baseline and periodic tests of lung function, as well as provide chest radiographs for all miners. The NIOSH recommendation as well as others were considered by an advisory panel on the elimination of pneumoconiosis empaneled by the secretary of labor (207). This committee issued findings and recommendations:

- Improved methods for inspection and enforcement of dust limits
- Enforcement of separate exposure limits for coal mine dust and silica that are lower than current levels
- Improved hazard surveillance
- Expanded health screening and surveillance to include lung function for all miners
- Improved and expanded training in dust control.

Until the next century, however, many retiring miners will have experienced dust exposure prior to 1972 and will therefore be at increased risk for disease. Also, concerns are periodically raised about whether the dust samples analyzed by the Mine Safety and Health Administration (MSHA) accurately reflect dust conditions in the mines (208,209). Thus the risk of disease remains uncertain and preventive interventions are critical.

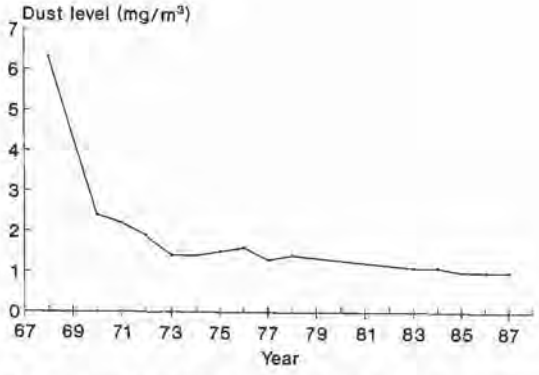


FIG. 13. Reported trends in dust levels for continuous miner operators 1968 to 1987. (Data taken from three reports.)

Working miners with evidence of CWP as identified under the NSCWP administered by NIOSH are entitled to work in a low-dust area within the mine (currently less than 1 mg/m^3 air) (210). 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 interpreted by trained readers, and results are sent to the miner and a personal physician if one is designated, but not to the employer. If a job transfer to a low-dust area of the mine is necessary, 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, as this is the only currently available preventive intervention.

REFERENCES

1. Gregory JC. Case of peculiar black infiltration of the whole lungs, resembling melanosis. *Edinburgh Med Surg J* 1831;36:391.
2. Medical Research Council of Great Britain. *Chronic pulmonary diseases in South Wales coal miners.* Special report series 243. London: Medical Research Council of Great Britain, 1942.
3. Medical Research Council of Great Britain. *Chronic pulmonary diseases in South Wales coal miners.* Special report series 244. London: Medical Research Council of Great Britain, 1943.
4. Smith BE. *Digging our own graves:* coal miners and the struggle over black lung diseases. Philadelphia: Temple University Press, 1987.
5. Federal coal mine health and safety act. Publ. L No. 91-173, 2917. 1969.
6. Federal mine safety and health act of 1977. Publ. L No. 91-173. Amended by Publ. L No 95-164, 101. 1977.
7. Weeks JL, Wagner GR. Compensation for occupational disease with multiple causes: the case of coal miners' respiratory diseases. *Am J Public Health* 1985;76:58–61.
8. Meiklejohn A. History of lung diseases of coal miners in Great Britain: part I, 1800–1875. *Br J Ind Med* 1951;8:127–137.
9. Meiklejohn A. History of lung diseases of coal miners in Great Britain: part II, 1875–1920. *Br J Ind Med* 1952;9:93–98.
10. Meiklejohn A. History of lung diseases of coal miners in Great Britain: part III, 1920–1952. *Br J Ind Med* 1952;9:208–229.
11. Fay JWJ, Rae S. The pneumoconiosis field research of the National Coal Board. *Ann Occup Hyg* 1959;1:149–161.
12. McBride WW, Pendergrass EG, Lieben J. Pneumoconiosis study of Pennsylvania anthracite miners. *J Occup Med* 1966;8:365–376.
13. Tokuhata CK, Dessauer P, Pendergrass EP, Hartman T, Digon E, Miller W. Pneumoconiosis among anthracite coal miners in Pennsylvania. *Am J Public Health* 1970;60:441–451.
14. Lieben J, Pendergrass E, McBride WW. Pneumoconiosis study in central Pennsylvanian coal miners. *J Occup Med* 1961;5:376–388.
15. Hyatt RE, Kistin AD, Mahan TK. Respiratory disease in southern West Virginia coal miners. *Am Rev Respir Dis* 1964;89:387–401.
16. McBride WW, Pendergrass E, Lieben J. Pneumoconiosis study of western Pennsylvania bituminous-coal miners. *J Occup Med* 1963;5:376–388.
17. Higgins ITT, Higgins MW, Lockshin MD, Canale N. Chronic respiratory disease in mining communities in Marion county, West Virginia. *Br J Ind Med* 1968;25:165–175.
18. Lainhart WS. Roentgenographic evidence of coal workers' pneumoconiosis in three areas in the United States. *J Occup Med* 1969;11: 399–408.
19. Attfield MD, Castellan RM. Epidemiological data on US coal miners' pneumoconiosis, 1960 to 1988. *Am J Public Health* 1992;82:964–970.
20. Butani SJ, Bartholomew AM. *Characterization of the 1986 coal mining workforce.* Bureau of Mines information circular IC 9192. Washington, DC: US Bureau of Mines, 1988.
21. Attfield MD, Althouse RB. Surveillance data on US coal miners' pneumoconiosis, 1970 to 1986. *Am J Public Health* 1992;82:971–977.
22. Enterline PE, Lainhart WS. The relationship between coal mining and chronic nonspecific respiratory disease. *Am J Public Health* 1967;57: 484–495.
23. Higgins ITT. Chronic respiratory disease in mining communities. *Ann N Y Acad Sci* 1972;200:197–210.
24. Higgins ITT, Cochrane AL. Chronic respiratory disease in a random sample of men and women in Rhondda Fach in 1958. *Br J Ind Med* 1961;18:93–102.
25. Higgins ITT, Oh MS, Whittaker DE. *Chronic respiratory disease in coal miners.* DHHS (NIOSH) publication 81-109. Washington, DC: US Department of Health and Human Services, 1981.
26. Enterline PE. Mortality rates among coal miners. *Am J Public Health* 1964;54:758–768.
27. Rockette H. *Mortality among coal miners by the UMWA health and retirement funds.* DHEW (NIOSH) publication 77-155. Washington, DC: US Department of Health, Education, and Welfare, 1977.
28. Liddell FDK. Mortality of British coal miners in 1961. *Br J Ind Med* 1973;30:1–14.
29. Ortmeyer CE, Costello J, Morgan WKC, Swecker S, Petersen MR. The mortality of Appalachian coal miners. *Arch Environ Health* 1974;29:67–72.
30. Miller BG, Jacobsen M. Dust exposure, pneumoconiosis, and mortality of coal miners. *Br J Ind Med* 1985;42:723–733.
31. Cochrane AL, Carpenter RG, Moore F, Thomas J. The mortality of miners and ex-miners in the Rhondda Fach. *Br J Ind Med* 1964;21: 38–45.
32. Cochrane AL. Relation between radiographic categories of coalworkers' pneumoconiosis and expectation of life. *Br J Ind Med* 1973;2: 532–534.
33. Cochrane AL, Haley TJL, Moore F, Hold D. The mortality of men in the Rhondda Fach, 1950–79. *Br J Ind Med* 1979;36:15–22.
34. Cochrane AL, Moore F. Preliminary results of a twenty-year follow-up of a random sample of an industrial town. *Br Med J* 1978;35: 411–412.
35. Cochrane AL, Moore F. A 20-year follow-up of men aged 55–64 including coal-miners and foundry workers in Staveley, Derbyshire. *Br J Ind Med* 1980;37:226–229.
36. Ortmeyer CE, Baier EJ. Life expectancy of Pennsylvania coal miners compensated for disability. *Arch Environ Health* 1973;27:227–230.
37. Meijers JMM, Swan GMH, Slangen JJM, van Vliet K, Sturmans F. Long-term mortality in miners with coal workers' pneumoconiosis in the Netherlands. *Am J Ind Med* 1991;19:43–50.
38. Goldman KP. Mortality of coal-miners from carcinoma of the lung. *Br J Ind Med* 1965;22:72–77.
39. Ames RG, Amandus H, Attfield M, Green FY, Vallyathan V. Does coal mine dust present a risk for lung cancer? A case-control study of U.S. coal miners. *Arch Environ Health* 1983;38:331–333.
40. Stocks P. On the death rates from cancer of the stomach and respiratory diseases in 1949–53 among coal miners and other residents in counties of England and Wales. *Br J Cancer* 1962;16:592–598.
41. Matolo NM, Klauber MR, Gorishek WM. High incidence of gastric carcinoma in a coal mining region. *Cancer* 1972;29:733
42. Swaen GMH, Meijers JMM, Slangen JJM. Risk of gastric cancer in pneumoconiotic coal miners and the effect of respiratory impairment. *Occup Environ Med* 1995;52:606–610.
43. Une H, Esaki H, Osajima K, Ikui H, Kodama K, Hatada K. A prospective study on mortality among Japanese coal miners. *Ind Health* 1995;33:67–76.
44. Ames RG. Gastric cancer in coal miners: some hypotheses for investigation. *J Soc Occup Med* 1982;32:73–81.
45. Meyer MB, Luk GD, Sotelo JM, Cohen BH, Menkes HA. Hypothesis: the role of the lung in stomach carcinogenesis. *Am Rev Respir Dis* 1980;121:887–892.
46. Jacobsen M, Rae S, Walton WH, Rogan JM. The relation between pneumoconiosis and dust exposure in British coal mines. In: Walton WH, ed. *Inhaled particles* III. Old Woking, England: Unwin Brothers, 1971:903–919.
47. Walton WH, Dodgson J, Hadden GG, Jacobsen M. The effect of quartz and other non-coal dusts in coalworkers' pneumoconiosis. In: Walton WH, ed. *Inhaled particles* IV, vol 2. Old Woking, England: Unwin Brothers, 1977:669–689.
48. Hurley JF, Burns J, Copland L, Dodgson J, Jacobsen M. Coalworkers' simple pneumoconiosis and exposure to dust at 10 British coalmines. *Br J Ind Med* 1982;39:120–127.

49. Hurley JF, Maclaren WM. *Dust-related risks of radiological changes in coalminers over a 40-year working life.* Report on work commissioned by NIOSH. Edinburgh, Scotland: Institute of Occupational Medicine, 1987.
50. Hurley JF, Alexander WP, Hazledine DJ, Jacobsen M, Maclaren WM. Exposure to respirable coalmine dust and incidence of progressive massive fibrosis. *Br J Ind Med* 1987;44:661–672.
51. Jacobsen M, Maclaren WM. Unusual pulmonary observations and exposure to coalmine dust: a case-control study. *Ann Occup Hyg* 1982;26:753–765.
52. Soutar CA, Collins HPR. Classification of progressive massive fibrosis of coalminers by type of radiographic appearance. *Br J Ind Med* 1984;41:334–339.
53. Jacobsen M, Burns J, Attfield MD. Smoking and coalworkers' simple pneumoconiosis. In: Walton WH, ed. *Inhaled particles* IV. Oxford: Pergamon Press, 1977;759–772.
54. Muir DCF, Burns J, Jacobsen M, Walton WH. Pneumoconiosis and chronic bronchitis. *Br J Ind Med* 1977;2:424–427.
55. Cochrane AL. The attack rate of progressive massive fibrosis. *Br J Ind Med* 1962;19:52–64.
56. McLintock JS, Rae S, Jacobsen M. The attack rate of progressive massive fibrosis in British miners. In: Walton WH, ed. *Inhaled particles* III. Old Woking: Unwin Brothers, 1971;933–952.
57. Maclaren WM, Hurley JF, Collins HPR, Cowie AJ. Factors associated with the development of progressive massive fibrosis in British coalminers: a case-control study. *Br J Ind Med* 1989;46:597–607.
58. Cockcroft AE, Wagner JC, Seal EME, Lyons JP, Campbell MJ. Irregular opacities in coalworkers' pneumoconiosis—correlation with pulmonary function and pathology. *Ann Occup Hyg* 1982;26:767–787.
59. Crawford NP, Bodsworth FL, Dodgson J. A study of the apparent anomalies between dust levels and pneumoconiosis at several British collieries. *Ann Occup Hyg* 1982;26:725–744.
60. Reisner MTR. Results of epidemiological studies of pneumoconiosis in West German coal mines. In: Walton WH, ed. *Inhaled particles* III. Old Woking, England: Unwin Brothers, 1971;921–931.
61. Reisner MTR, Robock K. Results of epidemiological, mineralogical, and cytotoxicogical studies on the pathogenicity of coal-mine dusts. In: Walton WH, ed. *Inhaled particles* IV. Oxford: Pergamon Press, 1977;703–716.
62. Breuer H, Reisner MTR. Criteria for long-term dust standards on the basis of personal dust exposure records. *Ann Occup Hyg* 1988;32: 523–527.
63. Attfield MD, Morring K. The derivation of estimated dust exposures for U.S. coal miners working before 1970. *Am Ind Hyg Assoc J* 1992; 53:248–255.
64. Attfield MD, Morring K. An investigation into the relationship between coal workers' pneumoconiosis and dust exposure in U.S. coal miners. *Am Ind Hyg Assoc J* 1992;53:486–492.
65. Attfield MD, Seixas NS. Prevalence of pneumoconiosis and its relationship to dust exposure in a cohort of U.S. bituminous coal miners and ex-miners. *Am J Ind Med* 1995;27:137–151.
66. Attfield M, Kuempel E, Wagner G. Exposure-response for coal workers' pneumoconiosis in underground coal miners: a discussion of issues and findings. *Ann Occup Hyg* 1997;41(suppl 1):341–345.
67. Soutar CA, Hurley JF. Relation between dust exposure and lung function in miners and ex-miners. *Br J Ind Med* 1986;43:307–320.
68. Hurley JF, Maclaren WM. Factors influencing the occurrence of progressive massive fibrosis (PMF) in miners and ex-miners. *Ann Occup Hyg* 1988;32:575–583.
69. Miller BG, Campbell SJ, Cowie HA, et al. *The natural history and implications of irregularly-shaped small shadows on coalminers' chest radiographs.* Edinburgh: Institute of Occupational Medicine, 1990.
70. Amandus HE, Lapp NL, Jacobson G, Reger RB. Significance of irregular small opacities in radiographs of coalminers in the USA. *Br J Ind Med* 1976;33:13–17.
71. Collins HPR, Dick JA, Bennett JG, et al. Irregularly shaped small shadows on chest radiographs, dust exposure, and lung function in coalworkers' pneumoconiosis. *Br J Ind Med* 1988;45:43–55.
72. Rae S, Walker DD, Attfield MD. Chronic bronchitis and dust exposure in British coalminers. In: Walton WH, ed. *Inhaled particles* III. Old Woking, England: Unwin Brothers, 1971;883–896.
73. Kibelstis JS, Morgan EJ, Reger R, Lapp NL, Seaton A, Morgan WKC. Prevalence of bronchitis and airway obstruction in American bituminous coal miners. *Am Rev Respir Dis* 1973;108:886–893.
74. Leigh J, Wiles AN, Glick M. Total population study of factors affecting chronic bronchitis prevalence in the coal mining industry of New South Wales, Australia. *Br J Ind Med* 1986;43:263–271.
75. Schmidt U. Dust and non-specific respiratory disorders in foundry workers and coal miners in the Rhine-Ruhr area. *Rev Inst Hyg Mines* 1979;34:70–76.
76. Carta P, Aru G, Barbieri MT, Avataneo G, Casula D. Dust exposure, respiratory symptoms, and longitudinal decline in lung function in young coal miners. *Occup Environ Med* 1996;53:312–319.
77. Rogan JM, Attfield MD, Jacobsen M, Rae S, Walker DD, Walton WH. Role of dust in the working environment in development of chronic bronchitis in British coal miners. *Br J Ind Med* 1973;30:217–226.
78. Hankinson JL, Reger RB, Fairman RP, Lapp NL, Morgan WKC. Factors influencing expiratory flow rates in coal miners. In: Walton WH, ed. *Inhaled particles* IV. Oxford: Pergamon Press, 1977;737–755.
79. Attfield MD, Hodous TK. Pulmonary function of U.S. coal miners related to dust exposure estimates. *Am Rev Respir Dis* 1992;14:605–609.
80. Leigh J, Wiles AN. Factors affecting prevalences of mucus hypersecretion and airflow obstruction in the coal industry of New South Wales, Australia. *Ann Occup Hyg* 1988;32(suppl 1):1186–1188.
81. Jain BL, Patrick JM. Ventilatory function in Nigerian coal miners. *Br J Ind Med* 1981;38:275–280.
82. Seixas NS, Robins TG, Attfield MD, Moulton LH. Exposure-response relationships for coal mine dust and obstructive lung disease following enactment of the Federal Coal Mine Health and Safety Act of 1969. *Am J Ind Med* 1992;21:715–734.
83. Nemery B, Veriter C, Brasseur L, Frans A. Impairment of ventilatory function and pulmonary gas exchange in non-smoking coalminers. *Lancet* 1987;2(8573):1427–1430.
84. Marine WM, Gurr D, Jacobsen M. Clinically important respiratory effects of dust exposure and smoking in British coal miners. *Am Rev Respir Dis* 1988;137:106–112.
85. Love RG, Miller BG. Longitudinal study of lung function in coal miners. *Thorax* 1982;37:193–197.
86. Attfield MD. Longitudinal decline in FEV_1 in United States coalminers. *Thorax* 1985;40:132–137.
87. Bates DV, Pham QT, Chau N, Pivoteau C, Dechoux J, Sadoul P. A longitudinal study of pulmonary function in coal miners in Lorraine, France. *Am J Ind Med* 1985;8:21–32.
88. Dimich-Ward H, Bates DV. Reanalysis of a longitudinal study of pulmonary function in coal miners in Lorraine, France. *Am J Ind Med* 1994;25:613–623.
89. Henneberger PK, Attfield MD. Coal mine dust exposure and spirometry in experienced miners. *Am J Respir Crit Care Med* 1996;153: 1560–1566.
90. Hodous TK, Hankinson JL. Prospective spirometric study of new coal miners. In: *Proceedings of the International Symposium on Pneumoconiosis, 1988.* Shenyang, PRC: Chinese Society of Preventive Medicine, 1990, 206–211.
91. Seixas NS, Robins TG, Attfield MD, Moulton LH. Longitudinal and cross sectional analyses of exposure to coal mine dust and pulmonary function in new miners. *Br J Ind Med* 1993;50:929–937.
92. Petsonk EL, Daniloff EM, Mannino DM, Wang ML, Short SL, Wagner GR. Airway responsiveness and job selection: a study in coal miners and non-mining controls. *Occup Environ Med* 1995;52:745–749.
93. Morgan WKC, Seaton A. *Occupational lung diseases,* 3rd ed. Philadelphia: WB Saunders, 1995.
94. Hurley JF, Soutar CA. Can exposure to coalmine dust cause a severe impairment of lung function? *Br J Ind Med* 1986;43:150–157.
95. Leigh J, Driscoll TR, Cole BD, Beck RW, Hull BP, Yang J. Quantitative relation between emphysema and lung mineral content in coalworkers. *Br J Ind Med* 1994;51:400–407.
96. Coggon D, Inskip H, Winter P, Pannett B. Contrasting geographical distribution of mortality from pneumoconiosis and chronic bronchitis and emphysema in British coal miners. *Occup Environ Med* 1995; 52:554–555.
97. Gamboa PM, Jáuregui I, Urrutia I, Antépara I, González G, Múgica V. Occupational asthma in a coal miner. *Thorax* 1996;51:867–868.
98. Kuempel ED, Stayner LT, Attfield MD, Buncher CR. Exposure-response analysis of mortality among coal miners in the United States. *Am J Ind Med* 1995;28:167–184.
99. Fairman RP, O'Brien RJ, Swecker S, Amandus HE, Shoub EP. Respiratory status of surface coal miners in the United States. *Arch Environ Health* 1977;32:211–215.

100. Amandus HE, Hanke W, Kullman G, Reger RB. A re-evaluation of radiological evidence from a study of U.S. strip coal miners. *Arch Environ Health* 1984;39:346–351.
101. Banks DE, Bauer MA, Castellan RM, Lapp NL. Silicosis in surface coalmine drillers. *Thorax* 1983;38:275–278.
102. Attfield MD, Vallyathan V, Green FHY. Radiographic appearances of small opacities and their correlation with pathology grading of macules, nodules, and dust burden in the lungs. *Ann Occu HYG* 1994; 38(suppl 1):783–789.
103. Vallyathan V, Brower PS, Green FHY, Attfield MD. Radiographic and pathologic correlation of coal workers' pneumoconiosis. *Am J Respir Crit Care Med* 1996;154:741–748.
104. Wagner GR, Attfield MD, Parker JE. Chest radiography in dust-exposed miners: promise and problems, potential and imperfections. In: Banks DE, ed. *Occupational medicine:* state of the art reviews, vol 8, no. 1. Philadelphia: Hanley and Belfus, 1993;127–141.
105. Weiss W. Smoking and pulmonary fibrosis. *J Occup Med* 1988;30: 33–39.
106. Castellan RM, Sanderson WT, Petersen MP. Prevalence of radiographic appearances of pneumoconiosis in an unexposed blue collar population. *Am Rev Respir Dis* 1985;131:684–686.
107. Blank PD, Gamsu G. Cigarette smoking and pneumoconiosis: structuring the debate (editorial). *Am J Ind Med* 1989;16:1–4.
108. Ducatman AM, Yang WN, Forman SA. "B-Readers" and asbestos medical surveillance. *J Occup Med* 1988;30:644–647.
109. Attfield MD, Althouse RB, Reger RB. An investigation of inter-reader variability among X-ray readers employed in the underground coal miner surveillance program. *Ann Am Conf Gov Ind Hyg* 1986;14: 401–409.
110. Bourbeau J, Ernest P. Between and within reader variability in the assessment of pleural abnormality using the ILO 1980 international classification of pneumoconiosis. *Am J Ind Med* 1988;14:537–543.
111. Remy-Jardin M, Degreef JM, Beuscart R, Voisin C, Remy J. Coal worker's pneumoconiosis: CT assessment in exposed workers and correlation with radiographic findings. *Radiology* 1990;177:363–371.
112. Akira M, Higashihara T, Yokoyama K, et al. Radiographic type p pneumoconiosis: high resolution CT. *Radiology* 1989;171:117–123.
113. Harkin TJ, McGuiness G, Goldring R, et al. Differentiation of the ILO boundary chest roentgenograph (0/1 to 1/0) in asbestosis by high-resolution computed tomography scan, alveolitis, and respiratory impairment. *J Occup Environ Med* 1996;38:46–52.
114. Remy-Jardin M, Remy J, Farre I, Marquette CH. Computed tomographic evaluation of silicosis and coal workers' pneumoconiosis. *Radiol Clin North Am* 1992;30:1155–1176.
115. Kanner RE, Barkman HW Jr, Rom WN, Taylor AT Jr. Gallium-67 citrate imaging in underground coal miners. *Am J Ind Med* 1985;8: 49–55.
116. Heppleston AG. Essential lesion of pneumoconiosis in Welsh coal workers. *J Pathol Bacterial* 1947;59:453–460.
117. Gough J, James WRL, Wentworth JE. A comparison of the radiological and pathological changes in coal workers' pneumoconiosis. *J Fac Radiol* 1949;1:28–60.
118. Kleinerman J, Green FHY, Laqueur W, et al. Pathology standards for coal workers' pneumoconiosis. *Arch Pathol Lab Med* 1979;103: 375–432.
119. Miller AA, Ramsden F. Carbon pneumoconiosis. *Br J Ind Med* 1961;18:103–113.
120. Watson AJ, Black J, Doig AT, Nagelschmidt G. Pneumoconiosis in carbon electrode makers. *Br J Ind Med* 1959;16:274–385.
121. Zahorski W. Pneumoconiosis dans l'industrie du graphite artificiel. In: *Proceedings of the XIII International Congress on Occupational Health.* New York: Book Craftsmen Associates, Inc., 1961;828–832.
122. Green FHY, Laqueur WA. Coal workers' pneumoconiosis. *Pathology* 1980;15:333–410.
123. International Labour Office. *International classification of radiographs of pneumoconiosis,* rev. ed. Occupational Safety and Health Series no. 22, rev. 80. Geneva: International Labour Office, 1980.
124. Wagner JC, Wusteman FS, Edwards JH, Hill RJ. The composition of massive lesions in coal miners. *Thorax* 1975;30:382–388.
125. Green FHY, Althouse R, Weber KC. Prevalence of silicosis at death in underground coal miners. *Am J Ind Med* 1989;16:605–615.
126. Rivers D, Wise ME, King EJ, Nagelschmidt G. Dust content, radiology, and pathology in simple pneumoconiosis of coalworkers. *Br J Ind Med* 1960;17:87–108.
127. Rossiter CM, Rivers D, Bergman C, Casswell C, Nagelschmidt G. Dust content, radiology, and pathology in simple pneumoconiosis of coal workers. In: Davies CN, ed. *Inhaled particles and vapours* II. Oxford: Pergamon, 1967;419–437.
128. Rossiter CM. Relation of lung dust content to radiological changes in coalworkers. *Ann NY Acad Sci* 1972;200:465–477.
129. Ruckley VA, Fernie JM, Chapman JS, et al. Comparison of radiographic appearances with associated pathology and lung dust content in a group of coalworkers. *Br J Ind Med* 1984;41:459–467.
130. Fernie JM, Ruckley VA. Coalworkers' pneumoconiosis: correlation between opacity profusion and number and type of dust lesions with special reference to opacity type. *Br J Ind Med* 1987;44:273–277.
131. Davis JMG, Chapman J, Collings P, et al. Variations in the histological patterns of the lesions of coal workers' pneumoconiosis in Britain and their relationship to lung dust content. *Am Rev Respir Dis* 1983; 128:118–124.
132. Douglas AN, Colline HPR, Fernie JM, Soutar CA. The relationship between radiographic and pathological appearances of progressive massive fibrosis. *Ann Occup Hyg* 1988;1:561–566.
133. Douglas AN, Robertson A, Chapman JS, Ruckley VA. Dust exposure, dust recovered from the lung, and associated pathology in a group of British coalminers. *Br J Ind Med* 1986;43:795–801.
134. Kuempel ED, O'Flaherty EJ, Stayner LT, Attfield MD, Green FHY, Vallyathan V. Relationships between lung dust burden, pathology, and lifetime exposure in an autopsy study of U.S. coal miners. *Ann Occup Hyg* 1997;41(suppl 1):384–389.
135. Ryder RC, Lyons JP, Campbell H, Gough J. Emphysema and coal workers' pneumoconiosis. *Br Med J* 1970;3:481–487.
136. Naeye RI, Mahon JK, Dellinger WS. Effects of smoking on lung structure of Appalachian coalminers. *Arch Environ Health* 1971;22:190–193.
137. Lamb D. A survey of emphysema in coal workers and the general population. *Proc R Soc Med* 1976;69:14.
138. Ruckley VA, Gauld SJ, Chapman JS, et al. Emphysema and dust exposure in a group of coal workers. *Am Rev Respir Dis* 1984;129: 528–532.
139. Leigh J, Outhred KG, McKenzie HI, Glick M, Wiles AN. Quantified pathology of emphysema, pneumoconiosis, and chronic bronchitis in coal workers. *Br J Ind Med* 1983;40:258–263.
140. Glick M, Outhred KG, McKenzie HI. Pneumoconiosis and respiratory disorders of coal mine workers in New South Wales, Australia. *Ann NY Acad Sci* 1972;200:316–334.
141. Leigh J, Outhred KG, McKenzie HI, Wiles AN. Multiple regression analysis of quantified aetiological, clinical and post-mortem pathological variables related to respiratory disease in coal workers. *Ann Occup Hyg* 1982;26:383–400.
142. Douglas AN, Lamb D, Ruckley VA. Bronchial gland dimensions in coalminers: influence of smoking and dust exposure. *Br J Ind Med* 1982;37:760–764.
143. Brighan KL. Role of the free radical in lung injury. *Chest* 1986;89: 859–863.
144. Johnson KJ, Fantone JC, Kaplan J, Ward PA. In vivo damage of rat lungs by oxygen metabolites. *J Clin Invest* 1981;68:1277–1288.
145. Martin WJ, Gadek JE, Hunninghake GW, Crystal RG. Oxidant injury of lung parenchymal cells. *J Clin Invest* 1981;67(4):983–993.
146. Kuroda M, Murakami K, Ishikawa Y. Role of hydroxyl radicals derived from granulocytes in lung injury induced by phorbol myristate acetate. *Am Rev Respir Dis* 1987;136:1435–1444.
147. Hubbard RC, Ogushi F, Fells GA, et al. Oxidants spontaneously released by alveolar macrophages of cigarette smokers can inactivate the active site of α-antitrypsin rendering it ineffective as an inhibitor of neutrophil elastase. *J Clin Invest* 1987;80:1289–1295.
148. Blackford J, Castranova V, Jones W, Dey R. Induction of the inducible nitric oxide synthase gene by intratracheal instillation of silica, coal, titanium dioxide, and carbonyl iron. *FASEB J* 1995;9:767.
149. Vallyathan V, Schwegler D, Reasor M, Stettler L, Green FHY. Comparative in vivo cytotoxicity and relative pathogenicity of mineral dusts. In: Dodgson J, McCallum RI, Bailey MR, Fisher DR, eds. *Inhaled particles* VI. Oxford: Pergamon, 1988;279–289.
150. Susskind H, Rom WN. Lung inflammation in coal miners assessed by uptake of ^{67}Ga citrate and clearance of inhaled ^{99m}Tc DTPA aerosol. *Am Rev Respir Dis* 1992;148:47–52.
151. Lebeder VV, Khrenkova TM, Goldenko NL. The formation of paramagnetic centers during crushing of coal. *Solid Fuels Chem* 1978;12: 117–119.

152. Dalal NS, Suryan MM, Vallyathan V, Green FHY, Jafari B, Wheeler R. Detection of reactive free radicals in fresh coal mine dust and their implication for pulmonary injury. *Ann Occup Hyg* 1989;33:79–84.
153. Kuhn DC, Demers LM. Influence of mineral dust surface chemistry on eicosanoid production by the alveolar macrophage. *J Toxicol Environ Health* 1992;35:39–50.
154. Castranova V, Bowman L, Reason MJ, Lewis T, Tucker J, Miles PR. The response of rat alveolar macrophages to chronic inhalation of coal dust and/or diesel exhaust. *Environ Res* 1985;36:405–419.
155. Brain JD. The effect of increased particles on the number of alveolar macrophages. In: Walton WH, ed. *Inhaled particles* III. Old Woking, England: Unwin Brothers, 1971;209–233.
156. Bingham E, Barkley W, Murthy R, Vassalo C. Investigation of alveolar macrophages from rats exposed to coal dust. In: Walton WH, ed. *Inhaled particles* IV. Oxford: Pergamon, 1977;543–550.
157. Bowden DH, Adamson IYR. Adaptive responses of the pulmonary macrophagic system to carbon. I. Kinetic studies. *Lab Invest* 1978;38:422–429.
158. Adamson IYR, Bowden DH. Adaptive responses of the macrophagic system to carbon. II. Morphologic studies. *Lab Invest* 1978;38: 430–438.
159. Kuhn DC, Standley CFS, Ayouby NEI, Demers LM. Effect of *in vivo* coal dust exposure on arachidonic acid metabolism in the rat alveolar macrophage. *J Toxicol Environ Health* 1990;29:157–168.
160. Wallaert B, Lassalle P, Fortin F. Superoxide anion production by alveolar inflammatory cells in simple pneumoconiosis and in progressive massive fibrosis of nonsmoking coal workers. *Am Rev Respir Dis* 1990;141:129–133.
161. Rom WN, Bitterman PB, Rennard SI, Cantin A, Crystal RG. Characterization of the lower respiratory tract inflammation of non-smoking individuals with interstitial lung disease associated with chronic inhalation of inorganic dusts. *Am Rev Respir Dis* 1987;136: 1429–1434.
162. Rom WN. Basic mechanisms leading to focal emphysema in coal workers' pneumoconiosis. *Environ Res* 1990;53:16–28.
163. Takemura T, Rom WN, Ferrans VJ, Crystal RG. Morphologic characterization of alveolar macrophages from subjects with occupational exposure to inorganic particles. *Am Rev Respir Dis* 1989;140: 1674–1685.
164. Rom WN. Relationship of inflammatory cell cytokines to disease severity in individuals with occupational inorganic dust exposure. *Am J Ind Med* 1991;19:15–27.
165. Lapp NL, Lewis D, Schwegler-Berry D, Castranova V, Abrons H, Kung M. Bronchoalveolar lavage in asymptomatic underground coal miners. 56th Meeting of American College of Chest Physicians, Toronto, Canada 1990(abstr).
166. Lapp NL, Weber SL, Vallyathan V, Castranova V, Shumaker J. Cytokine profiles in bronchoalveolar fluid of asymptomatic coal miners: natural defense mechanisms. *Am J Respir Crit Care Med* 1995; 151:A571
167. Lassalle P, Gosset P, Aerts C, et al. Alveolar macrophages secretory dysfunction in coal workers' pneumoconiosis. Comparison between simple pneumoconiosis and progressive massive fibrosis. In: Mossman BT, Begin RO, eds. *Effects of mineral dusts on cells*. Berlin: Springer-Verlag, 1984;65–71.
168. Borm PJA, Meijers JMM, Swaen GMH. Molecular epidemiology of coal workers' pneumoconiosis: application to risk assessment of oxidant and monokine generation by mineral dusts. *Exp Lung Res* 1990; 16:57–71.
169. Vanhee D, Gosset P, Marquette CH, et al. Secretion and mRNA expression of TNF-α and IL-6 in alveolar macrophages and in lung of pneumoconiotic patients. *Am Rev Respir Dis* 1993;147:906A.
170. Driscoll KE, Hassenbein DG, Carter JM, Kunkel SL, Quinlan TR, Mossman BT. TNF-α and increased chemokine expression in rat lung after particle exposure. *Toxicol Lett* 1995;82/82:483–489.
171. Hajjar KA, Hajjar DP, Silverstein RL, Nachman RL. Tumor necrosis factor-mediated release of platelet derived growth factor from endothelial cells. *J Exp Med* 1987;166:235–241.
172. Piguet PF, Collart MA, Grau GE, Sappino A, Vassili P. Requirement for tumor necrosis factor for development of silica-induced pulmonary fibrosis. *Nature* 1990;344:245–251.
173. Vanhee D, Gosset P, Wallaert B, Voisin C, Tonnel AB. Mechanisms of fibrosis in coal workers' pneumoconiosis; Increased production of platelet-derived growth factor, insulin-like growth factor Type I, and transforming growth factor beta and relationship to disease severity. *Am J Respir Crit Care Med* 1994;150:1049–1055.
174. Kelley J. Cytokines of the lung. *Am Rev Respir Dis* 1990;141:765–781.
175. Worth G. Emphysema in coal workers. *Am J Ind Med* 1984;6: 401–403.
176. Morgan WKC, Handelsman L, Kibelstis JS, Lapp NL, Reger R. Ventilatory capacity and lung volumes of US coal miners. *Arch Environ Health* 1974;28:182–189.
177. Rogan JM, Ashford JR, Chapman PJ, Duffield KP, Fay JWJ, Rae S. Pneumoconiosis and respiratory symptoms in miners at eight collieries. *Br Med J* 1961;1:1337–1342.
178. Hankinson JL, Reger RB, Morgan WKC. Maximal expiratory flows in coal miners. *Am Rev Respir Dis* 1977;116:175–180.
179. Cochrane AL, Higgins ITT, Thomas J. Pulmonary ventilatory functions of coalminers in various areas in relation to the x-ray category of pneumoconiosis. *Br J Prev Soc Med* 1961;15:1–11.
180. Morgan WKC, Lapp NL, Morgan EJ. The early detection of occupational lung disease. *Br J Dis Chest* 1974;68:75–85.
181. Morgan WKC. Hyperinflation of the lungs in coal miners. *Thorax* 1971;26:585–590.
182. Cotes JE, Field GB. Lung gas exchange in simple pneumoconiosis of coal workers. *Br J Ind Med* 1972;29:268–273.
183. Seaton A, Lapp NL, Morgan WKC. Relationship of pulmonary impairment in simple coalworkers' pneumoconiosis to type of radiologic opacity. *Br J Ind Med* 1972;29:50–55.
184. Ulmer WT, Reichel G. Functional impairment in coal workers' pneumoconiosis. *Ann NY Acad Sci* 1972;200:405–412.
185. Lapp NL, Seaton A. Pulmonary function in coal workers' pneumoconiosis. In: Key MM, Kew LE, Bundy M, eds. *Pulmonary reactions to coal dust*. New York: Academic Press, 1971;153–185.
186. Rasmussen DL, Laqueur WA, Futterman HD. Pulmonary impairment in Southern West Virginia coal miners. *Am Rev Respir Dis* 1968;98:658–667.
187. Ferris BG. Epidemiology standardization project: part II. *Am Rev Respir Dis* 1978;suppl 118:1–120.
188. U.S.Department of Labor. *Federal regulations:* parts 718,722,725, *726 and 727:* black lung benefits. Washington, DC: U.S. Department of Labor, 1991.
189. Lanese RR, Keller MD, Foley MF, Underwood EH. Differences in pulmonary function tests among whites, blacks, and american indians in a textile company. *J Occup Med* 1978;20:39–44.
190. Lapp NL, Amandus HE, Hall R, Morgan WKC. Lung volumes and flow rates in black and white subjects. *Thorax* 1974;29:185–188.
191. Rossiter CE, Weill H. Ethnic differences in lung function: evidence for proportional differences. *Int J Epidemiol* 1974;3:55–61.
192. Sorlie PD, Rogot E. Mortality status in the national longitudinal mortality study. *Am J Epidemiol* 1990;132:983–992.
193. Hankinson JL, Wagner GR. Medical screening using periodic spirometry for detection of chronic lung diseases. *Occup Med State Art Rev* 1993;8:353–362.
194. Eisen EA, Oliver LC, Christiani DC, Robins JM, Wegman DH. Effects of spirometry standards in two occupational cohorts. *Am Rev Respir Dis* 1985;132:120–124.
195. Crapo RO, Morris AH, Gardner RM. Reference spirometric values using techniques and equipment that meet the ATS recommendations. *Am Rev Respir Dis* 1981;123:659–664.
196. Astrand PO, Rhyming I. A nomogram for calculation of aerobic capacity (physical fitness) from pulse rate during submaximal work. *J Appl Physiol* 1954;7:218–221.
197. Hodgkin J. Pulmonary rehabilitation. *Clin Chest Med* 1990;11: 447–454.
198. American Thoracic Society. Pulmonary rehabilitation. American Thoracic Society position statement. *Am Rev Respir Dis* 1981;124:663–666.
199. Tiep BL. Long-term home oxygen therapy. *Clin Chest Med* 1990;11: 505–522.
200. Wilt JL, Banks DE, Weissman DN, et al. Reduction of lung dust burden in pneumoconiosis by whole-lung lavage. *J Occup Environ Med* 1996;38:619–624.
201. Jacobson M. Respirable dust in bituminous coal mines in the U.S. In: Walton WH, ed. *Inhaled particles* III. Old Woking, England: Unwin Brothers, 1971;903–917.
202. Parobeck PS, Jankowski RA. Assessment of the respirable dust levels

in the nation's underground and surface coal mining operations. *Am Ind Hyg Assoc J* 1979;40:910–915.
203. Watts WF. Respirable dust trends in coal mines with longwall or continuous miner sections. In: *Proceedings of the VIIth International Pneumoconiosis Conference,* Pittsburgh, August 1988. DHHS (NIOSH) publication 90-108. Washington, DC: DHHS, 1990;94–99.
204. Weeks JL. Characteristics of chronically dusty longwall mines in the U.S. In: *Proceedings of the VIIth International Pneumoconiosis Conference,* Pittsburgh, August 1988. DHHS (NIOSH) publication 90-108. Washington, DC: DHHS, 1990;76–80.
205. Key MM. Health standards and standard setting in the United States. *Ann NY Acad Sci* 1972;200:707–711.
206. National Institute for Occupational Safety and Health. *Criteria for a recommended standard:* occupational exposure to coal mine dust. Cincinnati, OH: National Institute for Occupational Safety and Health, 1995.
207. U.S.Department of Labor. *Report of the secretary of labor's advisory committee on the elimination of pneumoconiosis among coal mine workers.* Washington, DC: U.S. Department of Labor, 1996.
208. Boden LI, Gold M. The accuracy of self-reported regulatory data: the case of coal mine dust. *Am J Ind Med* 1984;6:427–440.
209. Mine Safety and Health Administration. *Report of the statistical task team of the coal mine respirable dust task group.* Washington DC: US Department of Labor, 1993.
210. Specifications for medical examinations of underground coal miners. *Federal Register* 1973;38:20076–20081.
211. *U.S. geological survey.* Washington, DC: U.S. Government Printing Office, 1975.